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NOTES ON PATHOLOGY.

NOTES

ON

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PREFATORY NOTE.

For some time past Students of Pathology in Edinburgh University have experienced the want of a text book which, while moderate in dimensions, could be relied upon to supply a full knowledge of the subject.

In the absence of such a book, the compilers of these "Notes" have endeavoured to place before the student a succinct, yet comprehensive, account of the science, in the hope that it may render his work easier and at the same time more congenial than many are accustomed to find it. The various sections have been dealt with very much in the order adopted by Professor Greenfield in his lectures, and the works of Coats, Ziegler, Payne, and Woodhead have been consulted on many points. Nothing original is claimed for the work beyond what is hoped will prove to be clearness and system in the method of treatment.

To further aid the student, the volume has been interleaved, in order to provide space for diagrams, and for any additional notes which may be added in the lecture room.

Edinburgh, *October*, 1889.

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PART I.

GENERAL PATHOLOGY.

RETROGRESSIVE METAMORPHOSES.

CLOUDY SWELLING

Is a term which was first applied by Virchow to a peculiar change in the essential tissue of certain organs, and which he described as " hypertrophy with a tendency to degeneration."

Cause.—It may be due to (1) High temperature (fevers), (2) Early inflammations, (3) Deranged nutrition, (4) Poisons (animal or vegetable), or (5) Burns.

It occurs in those parts of the body having the most complex functions, the commonest positions being the kidney, liver, and muscular substance of the heart.

Microscopic appearance.—The cells affected become enlarged and granular, irregular in shape, and easily detached from one another, thus showing that the cement substance between the cells is affected. Vacuoles appear in the cells, the nuclei enlarge but become less distinct, and in later stages the cells break down.

Naked eye characters.—As a result of these cell changes the organ appears swollen, but its vascularity is diminished owing to the swollen cells lessening the lumen of the vessels, and consequently it has a pale cloudy appearance. If the disease occurs superficially instead of " en masse " the surface becomes irregular.

Chemical Characters.—On adding acetic acid the granules disappear. They do not stain with osmic acid, nor are they affected by carmine or logwood dyes ; therefore the granules are not fatty in nature, nor are they composed of protoplasm.

Theories as to Nature of Cloudy Swelling. (*a*) It is often stated to be due to precipitation of albuminous particles in the cell, hence called "albuminoid degeneration." (*b*) Virchow thinks the cell takes up more nourishment than it can digest, and so the waste particles are left. (*c*) Greenfield believes that the apparent particles are not separate, but are only the nodal points of the cell network shown with increased distinctness, and probably caused by an alteration in density of certain parts of the cell substance.

Results.—If the irritant be removed the organ may return to normal, otherwise the process may terminate in fatty degeneration.

FATTY DEGENERATION AND INFILTRATION.

Fatty Degeneration as distinguished from Infiltration is a true conversion of the protoplasm of the cell. Although the distinction is quite obvious, yet the one condition frequently runs into the other. Thus, after food is taken, the fatty materials brought to the liver by the portal vein are deposited at the periphery of the lobules as droplets of fat; gradually these droplets get oxidized and disappear. If, however, the fat be deposited so rapidly that it cannot be entirely oxidized, what is known as "fatty infiltration" takes place, *i.e.*, an abnormal quantity of fat is deposited in the cell. But if the protoplasm itself, through loss of vitality or a decreased supply of oxygen, becomes converted into fatty material, then the condition of "fatty degeneration" results.

FATTY DEGENERATION.

The positions in which it occurs are unlimited. The most common sites are the liver, kidney, and heart. Speaking generally, it occurs under conditions of defective nutrition, and mainly affects the cells of tissues.

Naked eye appearance.—The organ is paler than normal and is easily broken down. The weight and specific gravity are diminished, and a piece of the organ may float in water. In the later stages it is wasted.

Microscopic Characters.—The cells are usually shrunken, and contain minute isolated oil globules which are strongly refracting. The nucleus shares in the degeneration as a rule, but is sometimes unaffected.

Causation.—I. *General* (1) Defective nutrition, in *(a)* old age, from imperfect assimilation; *(b)* inanition, *e.g.*, in cancer of the stomach; *(c)* wasting diseases, *e.g.*, phthisis; (2) Imperfect oxidation, *e.g.*, from poisons, especially phosphorus and alcohol. II. *Local* (1) diminished blood supply; (2) loss of function, *e.g.*, in muscle fibre.

Chemical Reactions.—The affected parts stain black with osmic acid, the outline of the oil globules being well marked; benzol, ether, or chloroform dissolves out the oil. Acetic acid has no effect.

Nature of the Change.—Three theories have been put forward. (1) A chemical change takes place in the proteids of the tissue by which they split up, leaving fat as one of their residues. (2) In all tissues there is a certain amount of fat chemically combined with the proteid element, and when this combination is severed the fat appears. (3) The fat accumulates through insufficient oxidation.

The first view is of most importance, as illustrated in the following facts :—Freshly made cheese contains between 80 and 90% of casein and a small percentage of fat. If kept for a time and allowed to ripen, the fat may increase up to 30°/₀, while the casein correspondingly decreases. The inference is obvious that the fat has increased at the expense of the proteid. Further, there is a substance "adipocere," which is produced in dead bodies when allowed to remain at a low temperature in water or damp soil. The muscles become tallowy, and compounds of palmitic and oleic acids with ammonia and lime salts are found in them. If you place proteids in the same condition they will undergo a similar decomposition.

Again, if animals are fed on an exclusively nitrogenous diet, the amount of fat in the body remains normal, and the females secrete milk plentifully, and with the normal proportion of cream.

Lastly, the experiments of Bauer on phosphorus poisoning afford a very striking proof. Phosphorus has the property of diminishing oxidation in the body, and causing proteids to break down. Bauer ascertained the amount of urea excreted by a dog, and then poisoned it by small doses of phosphorus. He found that the urea increased in quantity day by day until the dog died. After death the organs were found in a state of extreme fatty degeneration. Thus, part of the nitrogen derived from the decomposed proteids was excreted as urea, and part stored up as fat.

In fatty degeneration the function of the organ affected is carried on, though weakly, unless the condition be far advanced. The organ may return to normal when the degeneration has been slight ; otherwise caseation or even calcification may result.

FATTY INFILTRATION.

Consists in a deposit of fat in tissues which do not normally contain fat, or an increased deposit in tissues which normally contain some fat. The connective tissue corpuscles between the muscular fibres of the heart do not normally contain any oil ; should they do so, we get one form of "fatty heart."

Fatty infiltration does not interfere with the function of an organ to the extent that fatty degeneration does. The globules are much larger and usually occur singly in a cell ; rarely more than two are seen in the same cell. In this condition the organ may be increased in weight although the sp. gr. is less than normal.

Causation.—*(a)* Excessive consumption of fats or carbo-hydrates. *(b)* Defective oxidation, *e.g.*, in lung disease; *(c)* Certain nervous diseases, *e.g.*, insanity: *(d)* Constitutional conditions, *e.g.*, repeated parturition; *(e)* Fevers,—it frequently follows typhoid.

Adiposity is a great increase of fat in normal positions, and is a pathological condition often associated with the above.

CALCIFICATION.

This condition is an evidence of degeneration, and is often a sequel to fatty degeneration. It is one of the commonest evidences of impaired nutrition of tissue, and while frequently found in man, it is still more common in herbivora. It consists in the deposit of salts similar to those which exist in normal bone, *i.e.*, calcium carbonate and phosphate, magnesium carbonate and phosphate.

If we have an increased quantity of lime and magnesium salts in food, they do not cause an increase in bone: but if they be omitted, the bones are impaired, especially if the animal be young and growing. In physiological calcification these salts are in intimate combination with the tissue, but such is not the case in pathological calcification. Deposition usually takes place in a granular form, first between the cells, later in the cells. The granules are very minute, but when they run together they present a crystalloid appearance. These larger masses are highly refractive, and impart a hardness and whitish colour to the tissue. The condition leads to increased bulk, and is frequently associated with decayed vitality and senile changes. It tends to occur in the arterial system more than the venous, because the carbonic acid in the venous blood retains the salts in solution. In the arterial system the chief parts affected are the aorta, mitral valve, and the deep layers of the inner coat in large arteries; and it is associated with atheroma, affecting the newly formed material in that condition. It very rarely attacks the smaller arteries, and the capillary and venous systems are usually free.

The structures in which the process frequently takes place may be enumerated as follows:—cartilages of ribs and larynx; tendons and fibrous membranes; tumours and dead tissue; parts which have undergone caseation, *e.g.*, bronchial and mesenteric glands; old pus, as in npyema ~~emphysema~~; blood clots, the so-called vein stones or phleboliths; secretions brought up from the fauces; parasites, T. spiralis and Hydatids; fœtus in utero dying frequently becomes calcified (lithopædion).

It is supposed that the condition is due to the affinity of fatty acids for lime salts, the acids becoming oxidized after a time, leaving the salts as a deposit.

It may occur by a transference of salts from bones to other tissues. In rickets and osteomalacia lime salts have been found deposited in the liver and kidney—calcareous metastasis.

In calcification, as distinguished from ossification, the bony tissue shows no Haversian canals nor lacunae.

CASEATION

Usually follows fatty degeneration, and derives its name from the cheesy appearance of its product. The process takes place in tissues which have been cut off from their blood supply, such as a gumma resulting from endarteritis obliterans of a blood vessel, the area supplied by the vessel degenerating and caseation setting in. It is also well seen in an infarct as the result of embolism.

The conversion of a tissue into a cheesy mass may occur in one of three ways. (1) It may gradually assume as a whole a more and more homogeneous appearance, the cells fusing together and losing their nuclei, *e.g.*, in tuberculosis. (2) Detached homogeneous masses may first be formed which fuse together into one continuous mass. (3) The tissue may first dissolve and be replaced by granules and granular fibrils of fibrin; these then coalesce into a dense uniform mass, *e.g.*, in pneumonia.

Microscopic appearance.—One can distinguish the outlines of degenerated cells (devoid of nuclei) minute granules, fatty acids, oil globules, and cholesterin crystals. These last are transparent rhombic plates notched at the angles; they are soluble in ether, chloroform, and bile acids, also in hot alcohol, insoluble in cold alcohol. When gently heated with five parts of sulphuric acid and one part of water, the crystals turn carmine at the edges.

The ultimate condition of the caseous mass is either softening and liquefaction with absorption, or calcification.

SEROUS OR DROPSICAL DEGENERATION

Are terms applied to a process which very much resembles cloudy swelling. The degeneration chiefly affects epithelial cells, which imbibe fluid and become swollen in appearance. Vacuoles appear in the cells, the spherical vacuoles containing a clear liquid. The nucleus also swells up, and at length appears as a distended vesicle with clear contents. The condition is well seen in burns, when the deeper epithelial cells are observed to be chiefly affected. The change also takes place in liver cells.

Cause.—It seems to be a rapid absorption of fluid from the lymph.

MUCOID DEGENERATION.

There are two varieties of this degeneration. (1) Affecting cells and cell products (Mucoid degeneration proper). (2) Affecting connective tissue (Myxomatous degeneration.)

(1) A physiological example of **mucoid degeneration** is found in the salivary corpuscles. The mucoid degeneration of cells resembles that which takes place in normal secretion of mucus, but there is greater distension of the cells. The condition is well seen in catarrh of a mucous surface when the normal secretion is exaggerated. It is found in cysts, and in degenerative products of cells. The exudation stage in pneumonia is a beautiful example of the latter form.

(2) **Myxomatous Degeneration** in connective tissue. This not only occurs in connective tissue, but also in cartilage and bone. Mucoid connective tissue exists in the fœtus, forming a great part of the subcutaneous fat, which afterwards becomes "Wharton's jelly" of the umbilical cord.

Nature of the Change.—Instead of fibres of connective tissue with flattened cells we get branching cells with the interspaces filled with mucus. So in cartilage, the matrix becomes liquefied and the cells persist. Similar changes take place in bone. In some cases the cells also in connective tissue may undergo mucoid change—become "dropsical." As the tissue seems to return to its original embryonic form, it may be more properly called a "degradation" than a degeneration. Virchow calls it Metaplasia, *i.e.*, conversion of one type of tissue into another. Degeneration of the villi of the chorion is a good example, forming the "hydatid mole." Similar changes take place in cartilaginous tumours.

Chemical Reactions.—Mucin swells up in water, and is coagulated by acetic acid or alcohol.

COLLOID DEGENERATION.

This degeneration is allied to the former, but is almost limited to epithelial cells and their degenerated products. We have colloid material normally in the thyroid and prostate. When it increases in the thyroid, goître results. In the prostate, it occasionally forms masses which calcify and become calculi. It is often found in the kidney, forming small cysts the size of a pin's head, as in "granular contracted kidney."

Naked eye appearance.—The tissue is swollen, and has the consistence of half-set glue. The condition is well seen in colloid cancer where the new material appears like sago or tapioca. Very often the degenerated parts have a yellow colour.

Microscopic appearance.—The mass of the cell becomes swollen, more highly refractile, and infiltrated with some jelly-like material, the nucleus persisting. Later, the nucleus may disappear. As the cells degenerate they become more or less fused, forming masses of this colloid material.

Nature of the change.—Virchow states that a substance called *colloidin* has been separated. This is not a proteid, and differs from mucin in containing sulphur.

Chemical Reactions.—It is distinguished from mucoid degeneration by non-precipitation on adding alchohol or acetic acid. But, on the other hand, tannic acid coagulates it.

GLYCOGENIC DEGENERATION.

There is a degeneration found in the kidney, and to a less extent in the liver in diabetes. Its nature is not fully made out, but one variety is known as glycogenic degeneration. In the kidney the degenerative process takes place in Henle's looped tubes and the collecting tubules. It consists in a change in the epithelial cells, which become swollen, homogeneous looking, and hyaline, but at the same time the outlines of the cells are preserved. In some cases where the liver has been secured immediately after death, glycogen has been observed as particles in the cells. Whether this should be called glycogenic degeneration or not is doubtful.

DEGENERATIONS AFFECTING FORMED TISSUES.

In this group of degenerations the affected parts undergo a homogeneous swelling, becoming more translucent and losing their characteristic appearance. They are further characterized by an increased durability in the tissues, and are recognized by (1) micro-chemical reactions, (2) conditions of occurrence, and (3) the particular structures affected.

The group includes Vitreous, Hyaline, and Waxy Degenerations.

A.—VITREOUS DEGENERATION (Zenker's Degeneration).

The condition is seen in muscle which has been subjected to severe strain or injury It is also observed in cases of typhoid fever, and is then probably due to the inflammatory condition. The muscle becomes pale like that of a fish.

Microscopic Changes.—These are well seen in the abdominal muscles in typhoid. The fibres loose their striation, become homogeneous, and acquire a vitreous or glassy appearance. They are brittle and tend to fracture transversely.

Stains.—Dull pink with picro-carmine, very like the staining of colloid material.

Nature of Change.—There is diversity of opinion among authorities. Some say it is like colloid change. Cohnheim groups it under "Coagulative Necrosis" produced after death. Others say it is analagous to hyaline degeneration.

B.—HYALINE DEGENERATION.

Is a disease of connective tissue and muscular fibres in arterioles, affecting chiefly the arterioles of the brain, pia mater, spleen, and kidney, and also ganglionic cells. The affected tissues acquire a peculiar translucent appearance. The change is apt to occur in irregular distribution, frequently as localized thickenings. When the arteries are affected, the epithelium lining the vessels is usually normal, although the inner coat is swollen, and the lumen almost obliterated. The middle coat is usually uniformly affected. In the outer coat the changes are less seen in a typical form.

Causation.—It occurs in blood poisonings—animal, vegetable, or mineral—pyæmia and septicæmia, scarlet fever, and inflammations. In diphtheria, it involves the spleen and kidneys.

REACTIONS.

	Healthy tissue.	Hyaline.	Waxy.
Iodine,	Yellow (canary).	Yellow.	Mahogany brown.
Iodine and sulphuric acid,	Yellow.	Yellow.	Slaty blue.
Methyl violet,	Blue.	Blue (sometimes red).	Rose violet.

Hyaline tissues are not affected by acetic acid, nor coagulated by alchohol, which only causes a shrinking. Like all proteid substances, hyaline matter stains deeply with Millon's reagent.

Nature of Change.—It consists of a chemical change in the tissue elements, probably a simple combination of the tissues with some of the proteids of the blood, a kind of coagulation of fibrin. The change is most likely due to the localized action of some poison, probably to some ferment evolved by micro-organisms. Similar changes may be produced by dilute acids injected into the vessels of a newly dead body.

C.—WAXY DEGENERATION ("Amyloid," "Lardaceous").

Next to "fatty," this is the commonest of all degenerations, and is very widely spread. The tissues affected present a peculiar homogeneous swelling, and are more translucent than normal. There is an increase in size, weight, and specific gravity of the organ affected. Owing to the lumen of the vessels being narrowed, the organ is paler than normal.

Sites.—The organs most frequently affected are the liver, spleen, kidneys, intestine and lymphatic glands; while the tissue elements involved are chiefly connective tissue fibrils and basement membranes. The elements, in order of affection, are the connective tissue of the smaller arteries, arterioles, capillaries as they are given off from the arterioles, and at more advanced periods, basement membranes. The change begins in the middle coat of arteries and extends outwards. As a rule it is the delicate connective tissue fibrils lying between the muscle

cells which are first affected. This is a distinction between waxy and the hyaline condition. In the latter degeneration, both muscular and connective tissue elements are affected, but rarely do we find the muscle cells affected in waxy. The vessels become so thick that capillaries can be divided into three coats. Waxy material has a remarkable power of resisting subsequent degenerations, and it occupies a unique position in this respect.

Conditions of occurrence —According to the researches of Hilton Fagge, and those of more recent date carried on by Dr. Gibson, the condition occurs most frequently in chronic phthisis, to a less extent in chronic suppuration, and to a less extent still in syphilis. In a few cases it has been found in malignant disease, and following acute rheumatism.

Nature of the change—If we digest a waxy organ artificially, there is left behind a substance called "Lardacein." It is soluble in strong hydrochloric acid. If diluted with water syntonin is precipitated, thus showing the proteid nature of the substance. It is decomposed by strong sulphuric acid, giving rise to leucin and tyrosin. It is soluble in alkalies. Eosin stains it a bright red colour.

Some time ago a committeee of the Pathological Society analysed the ash of specimens from three healthy, and three lardaceous livers for potassium, sodium, phosphoric acid, and chlorine; and arrived at the following results:—

	Potassium.	Sodium.	Phosphoric Acid.	Chlorine.
Healthy Livers, average	·2821	·0948	·3295	·1073
Lardaceous ,, ,,	·1319	·1902	·1981	·1664

Since potassium and phosphoric acid predominate in the blood corpuscles and pus corpuscles, while sodium and chlorine predominate in blood serum, these figures show that a great loss of pus corpuscles (as in chronic suppuration) would produce a deficiency of those elements which are actually found to be lacking. But this does not account for the occurrence of the waxy degeneration apart from suppuration.

It is supposed to be a degenerative change in the tissue elements (probably caused by bacteria), by virtue of which they are enabled to take up certain proteid elements from the blood, the combination resulting in the waxy change (Greenfield).

There is no sufficient evidence to show that the waxy is a further stage of the hyaline change, although they often lie side by side. The condition may occur within four months of perfect health, for instance after a suppurating gun shot wound, though it usually takes a much longer time to develop.

Results.—The tissues may return to normal if the condition be not far advanced, and the cause removed. They may break down into a granular mass. Fatty degeneration, and even calcification may take place. The walls of the vessel are quite permeable to fluid, thus allowing an exosmosis of the waxy material.

Chemical Reactions.—See Hyaline degeneration.

LOCALIZED WAXY DEGENERATION.

We sometimes find in the tissues local patches which give the reactions of waxy change, particularly in inflamed muscle round an old abscess, in lymphatic glands, in tumours, especially malignant forms, in degenerated cartilage, and in some diseases of the eye. But the change is rare, and it is an open question whether there is not general waxy change in the body at the same time.

Corpora Amylacea—The term is applied to oval or flattened masses composed of concentric layers deposited round a central nucleus, which is not unfrequently the remains of a cell or blood crystal. They are found in the choroid plexus, membranes of the brain and spinal cord, prostate of old men, emphysematous lungs, cancerous tumours, and old blood clots. Virchow regards them as having the same composition as waxy material. Greenfield believes they are analogous to colloid casts. They sometimes become calcified.

Staining reactions.—Iodine, and Iodine with sulphuric acid give stains corresponding to the ordinary waxy change; but other reagents give stains which vary.

URATIC INFILTRATION.

In the condition of gout there are found deposits of acid urate of soda in the joints of the hands, feet, knee, &c. Similar deposits are found in tendons, fascia, subcutaneous connective tissue of the ears and eyes, connective tissue of the kidney, and on the valves of the right heart. The form of deposit is either in minute delicate needles or fine granules. It takes place first in the cells, and later between them, differing from calcification, where the deposit always takes place first in the matrix. In joints, the more superficial layers of the cartilage are affected, where it forms an incrustation, which, to the naked eye, looks very like white paint.

The condition is due to defective metabolism and oxidation. In gout, uric acid is found in the blood (Garrod), while there is a defective excretion of it by the kidneys. Galvani in one of his experiments ligatured the ureters of a bird, and found urates afterwards deposited in the tissues.

Chemical Reactions.—(1) Dilute acids dissolve the urates. (2) urine Murexide test:—evaporate ~~urates~~ with nitric acid, touch the residue with ammonia, and a purple colour is obtained.

PIGMENTARY DEGENERATION AND INFILTRATION.

Physiological pigment exists in the choroid, cutis, membranes associated with the nerve centres, hair, and muscle. Organs containing an

excess of pigment are not necessarily impaired either in structure or function, though the condition is usually associated with some disease (Payne).

Pathological pigmentation may be classified into I.—that which is elaborated within the body, and II.—that derived from without.

I. *(a)* **True degeneration.**—The best example of this form is seen in the condition of brown atrophy of the heart, when the normal pigment round the nucleus is markedly increased, while the muscle cell is atrophied. In the ganglionic cells of the sympathetic and central nervous system (locus niger) it is also found.

(b) **Abnormal quantity or distribution of normal pigment.** In Addison's disease there is an exaggeration of the normal pigment in the rete Malphigi of the skin. It chiefly affects the parts exposed to light. The mucous membranes, tongue, lips, and inside of the cheeks are also pigmented. Leprosy and pigmented moles of the skin are other examples. In melanotic sarcoma, and some forms of melanotic cancer, the pigment is chiefly in the cells, and is elaborated by them.

(c) **From the blood or Bile.**—In purpura, hæmorrhages are found beneath the skin; these give rise to pigmented patches, which, however, soon disappear owing to the absorption of the extravasated blood. But if we examine the part of a brain into which hæmorrhage has taken place, eight days after the injury, we may find pigmented crystals. These crystals have been known to persist for 25 years. How are they formed? When blood is effused, part of it is absorbed, some of the corpuscles are taken up by the lymphatics; the clot shrinks, becomes drier, and soon decays. The white corpuscles and fibrin undergo fatty degeneration. Some haemoglobin soaks into the surrounding tissues, and a certain amount remains in situ. As occurs in experiments, the hæmoglobin is supposed to split up into globin and hæmatin, the latter of which contains all the iron of hæmoglobin. The hæmatin splits up, and the iron is deposited in the tissues as the hydrated ferrous oxide, which appears as brown or blackish granules. The part which contains no iron goes to form hæmatoidin. The crystals of hæmatoidin appear as clinorhombic red prisms and ruby red needles.

In microscopic hæmorrhage into the lung, the active epithelial cells are the great factors in effecting storage of pigment. These cells are contractile, and have the power of taking up foreign bodies. The red corpuscles are taken up entire, but they soon get broken down in the body of the cell, and ultimately appear as small black pigmented granules (Langhans). In malarial fever the red corpuscles split up in the blood itself, and become transformed into masses of pigment; the liver, spleen, and brain get pigmented as a result of this. The condition is known as melanæmia, and is probably due to a vegetable parasite. In gangrene we get red, purple, green, and black pigment, due to a more direct transformation of the blood. After death from any cause, we frequently find parts of the liver, spleen, and intestines abnormally pigmented, and usually presenting a dark greenish colour, due to sulphuretted hydrogen escaping from the intestines and acting on the blood. In jaundice, the characteristic yellow hue is well known. It is due to the absorption of bile pigment into the system.

II. **Pigment derived from without.**—Pigment may be introduced by way of the skin, lungs, or alimentary canal. Absorption by the **skin** is not common; the best example is tattooing. By the **lungs.**—All who live in smoky towns have a great deal of pigment in their lungs, due to the carbon particles floating about the air being inhaled. Workers among coal, iron, vermilion, &c., have abnormally pigmented lungs. The pigment does not lie free, but is taken up by the air cells which transfer it to the lymphatics. Introduced by the **stomach.**—If silver salts be given medicinally for a considerable time, the metal gets locked up in the tissues in the form of albuminate, and by the action of light produces a dusky brown black appearance in the skin, which is permanent, a condition known as *argyria*. In workers among lead we often find a blue line round the gums. It is due to sulphuretted hydrogen in the sordes of the teeth transforming the lead into the sulphide.

ATROPHY.

In atrophy we have to do not with defective development, but with retrogressive change in parts originally well-formed and well-grown. There is decrease in size of the organ, with a diminution in the number and size of the cells, and therefore, although the organ still retains its physiological function, it does so to a less degree than normal. In old age there is what may be termed a physiological retrogression. Thus, a man ultimately dies, even when there is no question of disease, so soon as the advancing involution of his vital organs reaches a stage at which they can no longer efficiently perform their functions. The bones become lighter, more spongy, and altered in shape, and many of the organs undergo "senile atrophy." The generative organs in woman lose their functional power long before extreme old age. The thymus gland undergoes complete degeneration before the period of adolescence.

Pathological Atrophy may be considered under the following conditions:—

I. **Imperfect Nutrition.**—This may be due to defective blood supply, as in the partial obliteration of an important artery from pressure of a tumour. If we entirely cut off the blood supply from a part, death of the part necessarily follows, but if there is sufficient nutriment supplied to keep it alive and no more, then atrophy takes place. In cancer of the

stomach food cannot be normally assimilated, hence the patient's tissues gradually atrophy from starvation. The falling off of flesh in a phthisical patient is another example. Atrophy of an organ may take place because of diminished power of the tissue to nourish itself; this may occur as a result of excessive functional activity, thus following hypertrophy; then, it is associated with fatty degeneration. Starvation is another obvious cause of atrophy. The parts affected in order during starvation are, fat, glandular organs, muscle, the walls of the alimentary canal, bones, and lastly the nervous system. The same holds good in old age, but the heart is an exception,—it increases in size as age advances, due to increased functional demand.

II. **Total or partial loss of function.** -When the stomach no longer takes in food, either from absolute starvation or in cases of obstruction of the œsophagus, the organ becomes extremely wasted, being sometimes apparently not larger than a piece of intestine. This can only occur when life is prolonged by nourishment introduced through another channel, *e.g.* nutrient enemata. The other digestive organs suffer also, though in a less degree. When a limb is prevented from being used it atrophies; this is markedly seen after amputation of the leg immediately above the knee, when the thigh becomes very much reduced in size. Loss of function of a gland precedes its atrophy, *e.g.*, in obstruction of Stenson's duct, the parotid gland atrophies. The optic nerve atrophies on removal of the eye-ball. After the operation of colotomy, the intestine dwindles to a small cord.

III. **Pressure.** — A mediastinal tumour, or an aneurism growing from the aortic arch may press on the sternum or vertebrae, causing atrophy by obstructing their blood supply, and also by directly causing the death of the cells, against which they press. In hydrocephalus there is a great accumulation of fluid in the ventricles of the brain; the ventricles consequently get distended and effect pressure on the brain substance, which necessarily atrophies.

IV. **Trophoneurotic Atrophy.**—Muscles which are cut off from their motor nerves undergo slow wasting. Nerve-fibres cut off from their central connections (ganglionic cells) invariably waste. In that form of leprosy where anæsthesia is a marked feature, the bones atrophy, due to the affection of their nerve trunks.

NECROSIS

Is a term applied to death of a palpable part in the living body. **Somatic death** is death of the body as a whole. **Necrobiosis** means death of molecular elements. A tissue whose vitality is not very high, but merely balanced between life and death, requires but a slight cause to complete its decay (old age). According to the observations of Cohnheim, it may be formulated as a general law—the more complex the tissue, the easier is it killed. Parenchymatous elements (specialized tissues) die sooner than supporting tissues. Thus, brain tissue, renal epithelium, and intestinal epithelium die after two hours' deprivation of blood; while skin, bone, and connective tissues will live for twelve hours. If the blood supply be cut off from the spinal cord of an animal for one hour, we find total destruction of the grey substance, but the white part of the cord may revive.

CAUSES OF NECROSIS.

I. **Arrest of Nutrition.**—This may be produced by ligature of an artery, obstruction to a vessel by pressure, or by a thrombus, or spasm of the arteries. Raynaud's disease is one in which, from time to time, the extremities become cold and bloodless. Sometimes it reaches such a degree that the arterial supply is completely cut off, and gangrene sets in. It is supposed to be produced by a spasm of the arteries. Ergot probably acts in the same way. Venous obstruction rarely gives rise to necrosis, from the extensive collateral anastomosis. But if the capillaries are blocked, it may happen, *e.g.*, in intussusception of the bowel. In intense periostitis the periosteum may be separated from the bone by a layer of pus; the subjacent bone, being thus deprived of its blood supply, dies. Direct pressure to a part, by obstructing its blood supply, acts in a similar way. Thus, tight bandaging may cause necrosis, especially when the vitality of the tissue is very low. Again, bed sores probably result from direct pressure. A weak action of the heart predisposes to the condition. The rapid onset of sloughing bed sores, when the spinal cord is directly injured by fracture of the vertebrae (even in young persons previously healthy), and the remarkable fact of a unilateral injury to the cord becoming complicated with sloughing on the opposite side only, seem to point to some direct influence of the nerve centres. Bed sores always begin at spots where the skin is drawn tightly over bone, and which are in most cases anæsthetic as well as motionless, *e.g.*, the sacrum, angle of scapula, vertebral spine, elbows, &c.

II. **Cell Poisons.**—Any decomposing matters, such as ammoniacal urine, putrid pus, or other discharges, or dead tissue (all of which contain septic organisms), have a more or less deleterious effect on cell life. By inoculation of bacteria into the cornea, their necrotic effect has been demonstrated quite apart from inflammation. Necrosis of tissues is immediately produced by some animal poisons, such as that of snake bite, which seems to have a direct power of killing the tissues.

III. **Physico-chemical agents.**—Some chemical poisons act by impairing the blood supply, while others, such as sulphuric acid, act directly on the tissues. Necrosis of the jaw in match manufacturers is produced by the direct action of the fumes of phosphorus. The exposure of tissue to excessive heat or cold will cause necrosis, death taking place according

to the temperature and duration of action. If we expose a rabbit's ear to a temperature between 50° and 58° C., or – 16° C., it will cause necrosis in a few minutes. A temperature of from 46° to 48° C. produces inflammation in a few minutes, but if the irritation be kept up for some hours, necrosis sets in (Cohnheim).

IV. **Intense Inflammation.**—A very common issue of inflammation is the disintegration, solution, and liquefaction of the entire tissue. This is the case, for example, in all suppurations, to a greater or less extent. The exudation from inflamed mucous surfaces contains, as a rule, no fibrin, and does not coagulate. In dysentery, the mucous membrane of part of the intestine may slough off in the form of casts.

FORMS OF NECROSIS.

(a) **Anæmic.**—All causes which seriously interfere with the circulation of a part, and bring almost permanent arrest of its movement, or stasis, may lead to the death of the tissue. Such causes are thrombosis, embolism, closing of the vessels by disease or ligature, pressure on the tissue, inflammation, hæmorrhage, &c. The arrest of the circulation need not however be permanent, it is enough if it persists longer than a certain time—necrosis still ensues.

(b) **Coagulative.**—In coagulation of blood or lymph, the death of leucocytes is thought to liberate the fibrinoplastic substance, and the ferment which, with the fibrinogenous substance of the plasma, form fibrin. There occurs in the death of tissue cells a process which is in a certain sense the converse of this, and has been called "coagulative" or "hyaline" necrosis. There is coagulation which takes place within the cells, instead of outside them, as in coagulation of blood. The cell becomes penetrated by the lymph, which contains fibrinogen. With this, the fibrinoplastin of the cell combines, and coagulation results, producing a stiffness or rigidity which is analogous to rigor mortis (Weigert). The nucleus disappears in a few hours or days, then the whole cell becomes more homogeneous, and finally breaks up into debris.

(c) **Colliquative Necrosis** or softening, in which the affected tissue becomes as it were liquefied, is closely akin to coagulative necrosis. In each case the necrosed parts become saturated with liquid. Colliquation may thus either precede or follow upon coagulation. For example, the coagulation of the blood and formation of a clot is preceded by the liquefaction or solution of certain cells contained in the blood. A similar process may be observed in the case of blisters following upon burns.

Separation of a Necrotic Mass.—Any dead part of the body tends to be thrown off by the surrounding healthy tissue. This isolation is effected by inflammation. If the dead part be exposed to the air it is liable to become gangrenous. This is also the case when death is the result of organisms. In internal parts, the irritation set up by the foreign body produces a hyperæmic zone, and the tissue becomes infiltrated by leucocytes; and ultimately a band of fibrous tissue is formed, which may have the distinctness of a capsule. The walls of this capsule must have some power of absorption, as a great part (sometimes the whole) of the necrotic mass may be removed and only a fibrous scar left. The process of incapsulation is essentially the same around a parasite, a dead syphiloma, &c.

GANGRENE.

Is Necrosis which is accompanied or caused by putrefaction. We may therefore have a primary or secondary form.

(1) **Primary Gangrene** means necrosis caused by putrefaction as exemplified in cancrum oris, and in gangrene of the lung. It results from the direct introduction of bacteria into a part where there is imperfect nutrition, and a consequent impaired vitality of the tissue. Wounds are therfore frequently attacked.

(2) **Secondary Gangrene** exists when putrefaction has followed necrosis. No better example illustrative of this process could be given than the celebrated experiment of Chauveau. He twisted the spermatic cord in an animal, and it caused simple atrophy of the testicle. But, when he previously introduced septic fluids, the testicle underwent the process of gangrene.

Varieties.—I. **Dry Gangrene** or Mummification. Dry gangrene is commonly the result of necrosis in parts which are exposed to the air. Typical examples are afforded by the so-called senile gangrene of the extremities, especially the feet, where the circulation in old people is always more or less stagnant from feebleness of propulsion. When necrosis sets in, the affected part is engorged with blood, and thus exhibits a dark or livid coloration. After the death of the part, the coloring matter of the blood transudes and gives the tissues a dark red appearance. At the same time the tissues lose their moisture by evaporation. Dry gangrene may occur as a later stage of moist gangrene. Microscopically the tissues are seen to undergo a molecular disintegration and appear shrunken and withered. Bones, tendons, and cartilage resist the change for a long time. The odour of the part becomes very offensive, due to the evolution of various gases, such as ammonium sulphide, sulphuretted hydrogen, and phosphoretted hydrogen.

II. **Moist Gangrene** (Sphacelus)—If septic organisms reach a dead tissue which is rich in blood, dropsical or other fluids, or is inflamed, moist gangrene may set in. The changes are more rapid than in dry gangrene. The part becomes livid, green, then purple, and black patches make their appearance later. The epithelium undergoes rapid desquamation. Gases are frequently evolved during the process, giving rise to bullæ, and producing the so-called "gangrenous emphysema."

The bacteria produce ferments which are locally injurious to the part, but the ferments are also apt to be absorbed into the system, causing blood poisoning. Further, the bacteria themselves may enter the system and cause pyæmia or septicæmia. The parts in the neighbourhood of the gangrene are highly inflamed, and a line of demarcation is formed, separating the living tissue from the dead.

Noma or **Cancrum Oris** is an infectious form of gangrene, occurring in children, with rather an insidious progress. It involves the mouth and cheek, and may cause extensive destruction of these parts.

Hospital Gangrene is a condition affecting wounds, and is also transmissible from one person to another.

Inflammatory Gangrene consists in the death of a mass of tissue as a result of inflammation.

ALTERED CONDITIONS OF THE CIRCULATION.

HYPERÆMIA.

Is a local excess of blood which may be arterial or venous in nature.

ARTERIAL HYPERÆMIA (Active Congestion).

May be either idiopathic or collateral.

The **Idiopathic** form is due to relaxation of the muscular fibres of the arteries, brought about by paresis of the vasomotor nerves or by stimulation of the vaso-dilators ; or it may result from weakness, or paralysis of the muscular fibres themselves (such, for example, as is produced by heat or the alkaloid atropia).

Collateral Hyperæmia is merely the consequence of a diminished blood supply to some contiguous part. It takes place first in the neighbourhood of the anæmic part, but afterwards may extend to more remote organs.

VENOUS HYPERÆMIA (Passive Congestion)

Exists when there is obstruction to the outflow of blood from a part through the veins. This may be due to obliteration of the veins by compression, ligature, coagulation of the contained blood, or thickening and tortuosity of the walls (varicose veins). General venous congestion is a sequel of disease of the heart (mitral regurgitation especially), or lungs (emphysema).

Effects on part.—If the congestion be complete and suddenly produced the part is apt to die rapidly, as in strangulated hernia. If, however, it be chronic and incomplete, the following changes are then liable to occur.

(1) **In Large Veins.** The vessels become greatly distended, and a local inflammatory change takes place in the walls, whereby they become thickened; and, as the inflammation extends along the walls, they also become elongated and tortuous (varicose). The lumen of the veins being thus greatly increased, it is evident that the valves will be rendered incompetent; and where the condition is aggravated by the action of gravity (as in the legs), the blood may clot, or the skin over the veins may ulcerate, constituting varicose ulcer. Sometimes the ulceration eats into the vessels, and causes serious haemorrhage.

(2) **The Small Veins and Capillaries** may become distended to several times their normal calibre, and, if near the surface, they are apt to be thrown into folds. The walls become impaired, leading to thickening and fatty degeneration. Rupture of the capillaries may take place, followed by haemorrhage.

(3) **The Tissue Elements** undergo degenerative processes on account of imperfect oxidation. The cells lying around the vessels become atrophied from pressure. This is well seen in a congested liver, where the cells in the central zone of the lobules may disappear. If haemorrhages take place, the tissues may appear pigmented. The organ affected is swollen, firmer and more vascular, and owing to the difference in the relative pressure within and without the capillaries and venules, dropsy is apt to supervene.

DROPSY (HYDROPS)

Consists in accumulations of serum in the tissue spaces or serous cavities. The condition receives different names as it occurs in different tissues :—

Of the integumentary system—Anasarca ; connective tissue generally—Œdema ; peritoneal cavity—Ascites ; pleural cavity—Hydrothorax ; pericardial cavity—Hydropericardium ; tunica vaginalis—Hydrocele ; ventricles of the brain—Hydrocephalus.

Nature of the fluid.—Dropsical fluid is simply diluted blood serum, containing less albumen and white corpuscles. It is clear and watery, and has a pale straw colour. It does not coagulate spontaneously, but if blood or fibrin ferment be added, coagulation takes place. If the dropsy has continued for some time, it will contain a relatively greater quantity of albumen. In serous cavities, it contains a larger proportion of albumen than in other situations. In *inflammatory* dropsy the fluid is rich in albumen, and contains fibrin, so that it coagulates almost as readily as blood. When **Jaundice** is associated with dropsy, the dropsical fluid is yellow in colour from bile staining. In *renal* dropsy urea is present, and in *diabetes* it contains glucose. On standing, dropsical fluid becomes concentrated, its proteids undergo fatty degeneration, and hence we find cholesterin crystals.

Causes of dropsy.—It is due either to increased transudation from, or diminished absorption by the vessels. Physiological absorption and transudation work reciprocally in maintaining an equable supply of lymph to the tissues. In morbid conditions this balance is disturbed; and, from the observations of many authorities, it appears that the chief disturbing factor is increased transudation from the vessels to the tissues.

I. **Diminished absorption** may be caused by obstruction of the (*a*) lymphatics, or of the (*b*) veins.

(*a*) If the *lymphatic vessels* of a limb be tied as completely as possible, no œdema is produced. This negative result may be due to the fact that it is impossible to entirely stop the lymph circulation owing to the number of anastomotic vessels, and also that new channels are rapidly developed. In addition, the veins possess the power of absorbing fluid from the tissues, and they may further aid compensation by a hyperdevelopment of this absorbent function. If the thoracic duct be entirely obstructed, as a rule no dropsy is produced. When, however, there is wide-spread obstruction of a number of the lymphatic vessels, as in elephantiasis, dropsy makes its appearance.

(*b*) Obstruction of the *veins* is a more important cause of dropsy. Obstruction of the portal vein usually gives rise to ascites. In cases of accidental ligature of the femoral vein in the human subject, we get œdema of the lower limb; although, in experiments on tying the same vein in a dog, no such result was obtained. This anomaly may be explained by the fact that the tissues of the animal operated on were probably more healthy. Under normal circumstances the veins act as absorbents of serum from the tissues, but during their obstruction it is evident they will cease to so act, and as the lymphatics are inadequate to carry off the excess of transuded fluid, dropsy supervenes.

II. **Increased Transudation.**—(*a*) There is evidence that in dropsy there is a greatly increased outflow of lymph through the lymphatic vessels; the very minute interfascicular spaces in the connective tissue become dilated into considerable cavities.

(*b*) As discussed under "Venous hyperæmia," increased transudation of lymph takes place in cases of obstruction to the circulation in veins. Arterial hyperæmia only acts as a cause of lymph transudation if the vitality of the arterial wall be impaired, as, *e.g.*, after nerve section.

(*c*) Changes in the Vascular Wall.—A very slight degree of local inflammation, causing damage to minute vessels, produces local dropsy. In famines, those suffering from starvation are liable to dropsy, which probably results from diminished nutrition of the vessel walls. Dropsy usually occurs in general cachexia, anæmia, and in chronic heart disease.

(*d*) Conditions of the Blood.—In cancer, phthisis, and Bright's disease, the blood deteriorates in quality; it becomes more watery, and contains less albumen. As weak albuminous solutions diffuse through dead animal membranes more easily than strong solutions, and in proportion to their lesser specific gravity, it was believed that dropsy occuring under the above circumstances was due to the watery condition of the blood. But according to the results arrived at through the experiments carried on by Cohnheim and Lichtheim, the walls of the capillaries do not allow of filtration like a dead membrane, for a watery state of the blood does not of itself produce dropsy; but it acts as a powerful concurrent cause along with venous hyperæmia. Therefore cancer, phthsis, and Bright's disease may be classed among those conditions which alter the nature of the filter (vessel wall).

(*e*) Nervous influence has a great deal to do with dropsy. It probably acts by paralysing the vasomotor nerves, and causing hyperæmia, which in combination with some other factor leads to effusion. In affections of the fifth nerve, œdema of the face is sometimes observed. In diseased conditions of the spinal cord, dropsy in the lower limbs is readily produced; it is most frequently seen in infantile paralysis.

(*f*) The action of Poisons.—Atropia diminishes the transudation of lymph, while arsenic frequently causes dropsy. Whether the actions of these drugs are on the blood or vessels, or on both, is rather obscure.

MECHANICAL ANÆMIA (LOCAL ANÆMIA)

Occurs whenever the blood supply to a tissue is partially or entirely cut off. If an artery be obstructed, there will necessarily be temporary anæmia of the part it supplies; but if it is not a terminal branch, and the neighbouring arteries be healthy, there is at once set up a sufficient collateral anastomosis which compensates for the affected vessel. Should, however, the neighbouring vessels be blocked or diseased, so that they cannot undergo dilatation, or if there is a weak action of the heart, whereby the propelling force is not sufficiently strong to produce dilatation of these vessels, the part will remain anæmic, and ultimately undergo local death or necrosis. The same condition holds with regard to terminal or end-arteries, such as the splenic vessels, the superior mesenteric artery, the coronary branches, and the central artery of the retina.

Causes of Obstruction.—(1) Arterial thrombosis or embolism; (2) Arterial obstruction through inflammation of the inner coat, *e.g.*, enderarteritis obliterans; (3) Arterial spasm; (4) Pressure from without, *e.g.*, tumour.

Ischæmia is the term applied when the obstruction is only partial. It frequently results from atheroma, and sometimes produces atrophy.

EMBOLISM.

Denotes the plugging of a blood vessel by the impaction of a ma

Increased Transudation

1 Increased outflow of lymph from lymphatic vessels

2 Increased press. of [illegible] from venous [illegible] obstruction [illegible]

3 Changes in vascular walls due to [illegible] nutrition etc

4 Altered condition of blood - more watery c̄ less albumen

5 Nervous influence - paralysis of [illegible] nerves causing hyperaemia

6 Action of poisons eg. arsenic

brought to it by the blood. This process can only take place in the systemic or pulmonary arteries, in the branches of the portal vein, and to a less extent in the capillaries.

The obstructive mass or *embolus* may be (1) part of a blood clot (the commonest cause) : (2) foreign body ; (3) oil globules ; (4) bacteria ; (5) air or gas ; (6) new growths, *e.g.*, cancer. It is obvious under what conditions these masses will produce embolism.

It was pointed out under "local anæmia," that when a terminal artery was blocked, necrosis resulted in the part supplied by the vessel. But before death occurs, various changes take place ; the area becomes engorged, and even diffuse hæmorrhage may occur. This constitutes a "hæmorrhagic infarction," and is only found in the lungs, spleen, kidneys, liver, retina, and rarely in the intestinal canal.

Characters of Infarcts.—Infarcts are mostly pyramidal in shape, and usually occur towards the surface of an organ, the base of the mass being the more superficial. The wedge shaped area corresponds to the part supplied by the vessel.

As the artery is blocked we might expect to find the tissue anæmic. But this is not so, or at least the anæmia is only transient ; on the other hand, the part becomes filled with blood. Whence does this blood come? It can only be derived from two sources, either by regurgitation from the veins, or by slow infiltration from capillaries which communicate with the arteries of adjacent territories. For some time Cohnheim held that the blood came as a reflux from the corresponding branch of the vein, but recent experiments entirely contradict this view. Litten tied a terminal branch of the renal artery, and connected a manometer with the corresponding vein, so that if the condition was due to a venous reflux there would be a corresponding rise in the venous blood pressure, but no such rise was observed. He further ligatured the vein as well as the artery, and still hæmorrhage took place. But on stripping off the capsule of the kidney and tying the ureter, in addition to ligaturing the artery, so that no capillary anastomosis might take place, he found no reflux Hence Litten concluded that the hyperæmia following arterial obstruction is due to the collateral arterial supply, although regurgitation from the veins may occur to aid the condition.

After a time the infarct becomes decolorised, its central part turns yellow and crumbles, or may break down into a pulpy mass resembling pus ; but suppuration does not occur unless the embolus has been septic. The paleness of the infarct is said to be due to fatty degeneration, which extends until the whole mass becomes caseous. In the kidneys the infarcts are usually very pale, while in the lungs (pulmonary apoplexy) they are always more or less red in colour. This is probably due to the fact that infarction in the latter position is usually associated with advanced cardiac disease, so that death takes place before the condition becomes quite completed. In pulmonary embolism there is no caseation, because the blood from the bronchial artery and vein keep up the nutrition. More typical examples of infarcts are seen in the spleen and liver.

The neighbouring blood vessels, receiving an excess of blood, dilate, so that a hyperæmic zone is found in the outer part of the infarct, the process ending in the formation of a thin capsule of fibrous tissue. If the condition be left for a considerable time, the mass disintegrates and is absorbed, leaving only a depressed fibrous scar to mark its position.

In solid organs the obstructed vessel is probably seldom opened, but in the brain and other positions where the vessels are not supported by solid tissue, this may occur especially in small capillaries. In such a case the vessel wall is left seriously damaged, so that a local dilatation or aneurism may be formed.

FORMS OF EMBOLISM.

(1) **Septic.**—Should the obstructive mass brought by the blood contain septic organisms derived from a wound or elsewhere, it will not only produce a degree of infarction as above, but will also excite active inflammation, and hence pyæmic abscess results.

(2) **Fat embolism** most frequently affects the pulmonary capillaries, and is either in the form of globules or long masses. The fat is usually derived from the bone marrow, and gains access to the vessels in cases of severe fractures or osteomyelitis; but fat may also enter the blood after contusions of subcutaneous fat. It also takes place in diabetes, where the sugar in the blood becomes transformed into fat; also in glanders and cancer.

(3) **Air embolism.**—In accidents, or during operations on the large veins of the neck, air may pass into a vessel and produce a fatal occurrence. It was at one time thought the evil effects resulted from air bubbles obstructing the pulmonary capillaries, but they are more probably due to accumulation of air in the right ventricle, which contracts ineffectually upon the air, and does not propel the blood.

(4) **Parasitic embolism.**—This condition, although common in certain animals, rarely, if ever, affects man.

(5) **Miliary** or **multiple embolism.**—In ulcerative endocarditis small vegetative growths become detached, and are carried by the blood stream to distant parts, and may give rise simultaneously to affections of the brain, spleen, kidney, &c. The effects vary with the size of the vessels affected, and on the presence or absence of bacteria.

(6) **Secondary** or **recurrent embolism.** — After an infarction has existed in the lung for some time, there may form a thrombus in the corresponding vein. If part of this be carried to the left side of the heart, it will reach some distal artery and give rise to obstruction.

Hæmorrhage into a bronchus may produce an appearance similar to a pulmonary infarct, because the bronchi are distributed in a similar way to the pulmonary arteries. But in the former case by the action of respiration, the blood would be carried in and out of the air cells, and hence the patch would not be so localized. Further, if the pulmonary artery corresponding to an infarct be examined, we will find an embolic plug.

THROMBOSIS.

The term means coagulation of blood in the vessels or the heart during life. It does not include extravascular coagulation.

The conditions which favour the occurrence of thrombosis are, (1) Irregularities of the heart or vessel wall, *e.g.*, vegetations on the heart valves, and atheroma. (2) Anything which tends to retard the circulation of the blood.as in the case of aneurism, defective force of the heart, varicose veins, &c. (3) Altered conditions of the blood, *e.g.*, acute rheumatism, when there is an excess of fibrin forming elements.

SITES OF OCCURRENCE.

(1) **Heart.** The most common positions in order of frequency are right auricular appendix, apex of right ventricle, apex of left ventricle, the left auricle (when it is usually associated with mitral stenosis), and on the mitral and aortic valves. (2) **Arteries.** It occurs less frequently in arteries than in veins because the circulation in the former is more active. In the large vessels atheroma predisposes to the condition, while in the small vessels mechanical injury or syphilitic endarteritis frequently give riseto it. (3) **Veins.** Thrombosis occurs most frequently in the veins, especially where there are plexuses. The process commonly begins in the pouches of the valves.

Having once started, a thrombus may continue to grow almost indefinitely, as it acts as a foreign body, and therefore constantly tends to coagulate the blood which comes in contact with it. A thrombus has been known to spread from a small vein in the leg into the inferior vena cava.

Varieties of Thrombi—(1) **Red Thrombi** chiefly consist of red corpuscles with no fibrin filaments and only a few leucocytes. The corpuscles become fused and show a reddish brown colour. These thrombi are formed when the blood clots rapidly "en masse." They are soft, and resemble clots formed outside the body. (2) **Pale Thrombi** are made up of white blood corpuscles with very few red. They are distinctly fibrinous, and present a hyaline homogeneous character not at all like a blood clot. Pale thrombi are dirty white in appearance, firm in consistence, and may be stringy or laminated. They are formed more slowly than the red thrombi, and while the blood is moving more actively.

If blood clots are formed after death, ("post mortem clots") they are always distinctly red in colour, present no vital changes, such as adhesions, and are very soft in consistence.

Method of Formation.—Before the discovery of a third corpuscular element ("blood plates") in the blood, it was thought that coagulation took place as it does outside of the body—the white corpuscles adhering to the rough surface and evolving fibrin ferment, in which red corpuscles were entangled. But the factors in producing thrombosis are more probably the blood plates; these are flat colourless discs, much smaller and fewer in number than the red corpuscles, and run in the axial stream. When the blood stream comes in contact with an irregularity on the vessel wall, these plates fall out of the axial stream and adhere to the rough surface, and form a hyaline homogeneous mass which constitutes the nucleus of the thrombus. The lamination of the clot is due to the laying down of successive layers at different periods, especially when the blood stream is slow.

Changes in Thrombus.—(1) A thrombus which plugs a vessel frequently contracts, and thus allows the vessel to become pervious. (2) It may undergo organization, *i.e.*, be replaced by connective tissue which unites it to the vessel wall (v. sub. Ligature of Arteries). (3) Fatty degeneration sometimes sets in, which may be followed by caseation or calcification. (4) The thrombus may soften and break down into a material like pus—"suppuration of the clot." This is usually the result of bacterial action. (5) The clot may become detached and give rise to embolism. (6) It may extend.

INFLAMMATION.

Inflammation may be defined as "the reaction of tissues and vessels to irritation."

Causes of Inflammation.—The irritants which produce inflammation may be grouped as (1) *Mechanical, e.g.*, traumatism; (2) *Physical, e.g.*, heat or cold; (3) *Chemical, e.g.*, chloride of zinc; (4) *Organismal*; (5) *Reflex, e.g.*, orchitis following urethral calculus.

There are four "cardinal signs" of inflammation, viz., redness, swelling, heat, and pain. To these may be added a fifth, viz., impairment or arrest of the function of the inflamed part. The above signs though usually present, are none of them essential to the process of inflammation. Redness and heat are signs of arterial hyperæmia; swelling depends on vascular fulness and exudation, while pain is the result of pressure or other influence on the sensory nerves.

1.—PHENOMENA OF INFLAMMATION IN VASCULAR TISSUES.

Experiments have been made on cold blooded animals, chiefly as the changes take place more slowly than in warm blooded animals. If

the omentum of a frog be exposed and an irritant applied, the following changes are observed to take place in order :—

(I.) **Active hyperæmia** of the vessels. The arteries first dilate, then the veins, and lastly the capillaries. The blood stream becomes faster, pulsation may extend into the capillaries, and the axial and peripheral streams become mixed. Simultaneously with these changes there is increased transudation of serum from the blood.

(II.) **Stasis.**—The blood stream first begins to slow in the venules, causing the axial stream to reappear. The slowing process gradually extends to the capillaries, and thence to the arteries, producing almost complete arrest of the blood flow (stasis). When stasis occurs the blood corpuscles again become mixed. **Theories as to causation.**—(1) Damage to vessel wall leading to adhesion of the white corpuscles to the wall, which acts as a foreign body (Lister). (2) Loss of elasticity in tissues surrounding the capillaries, involving loss of the natural stimulus to the propulsion of blood through these capillaries (Landerer). (3) Inability of the veins to carry off excess of blood, and consequent reaction.

III. The vessels become varicose and irregularly dilated, resulting from damage to their walls. **Diapedesis** of the white corpuscles take place; they accumulate in masses at the sides of the vessels, and by virtue of their amœboid movement escape into the surrounding tissues. The emigration of leucocytes may occur in from one to three hours after the application of the irritant, and is first observed to take place in the small veins; diapedesis does not take place through the capillaries, but only where there is a basement membrane. Endothelial plates may become loosened, especially in the veins. Red corpuscles escape from the capillaries, and even rupture of the minute vessels may take place.

(IV.) **Tissue Changes** take place subsequent to the vascular changes. The endothelial cells of the omentum enlarge, become more granular, and division of their nuclei takes place. Some of the cells become detached from their connective tissue basis, and may exhibit amœboid movement. At a later stage the connective elements proliferate, and undergo retrogressive changes. These changes are in great part due to increased nutriment, derived from the exudation. As the tissues lose their normal tone, by the action of the irritant on their nerves, they effect less pressure on the blood vessels, and hence this acts as a powerful factor in permitting of increased leucocyte emigration and lymph transudation; for, the more solid an organ is in which inflammation takes place, the less the exudation.

For the purpose of ascertaining the **essential phenomena of inflammation**, Cohnheim made the following experiments on the tongue of frogs :—

(1) The tongue was exposed, and at a point on the side, simple irritation was produced by means of pincers. In the vicinity of the

irritation, hyperæmia was excited, which spread throughout the whole tongue. This was followed by stasis gradually setting in. During the whole process no diapedesis nor increased exudation of lymph took place. But as the condition only lasted for about two hours, it may be said the time was too short for all the necessary phenomena of inflammation to take place.

(2) Cohnheim therefore took another animal, and scratched its tongue all over the surface, which resulted in the same changes as in the first experiment, only the hyperæmia was more marked, and the stasis lasted for a whole day.

(3) This time Cohnheim applied a caustic, which produced active hyperæmia throughout the tongue as in the case of the mechanical irritation. But at the seat where the caustic was applied there was a certain amount of destruction of the tissues, coagulation taking place in the neighbouring capillaries; beyond these vessels the hyperæmia gradually subsided and disappeared. In the area of irritation the vessels remained somewhat dilated, and the blood current gradually slowed. The stagnation of the circulation was such, that after five or six hours the individual corpuscles could be made out. There next occurred a period of reaction, which lasted from six to eight hours. The vessels in the vicinity became dilated, the stream in the dilated arteries being slower; at the same time diapedesis and transudation of lymph took place. The extravasation spread until it became general, and the whole tissue was filled with leucocytes and distended with lymph. The inflammation extended further out, so that at a considerable distance the vessels were markedly dilated, and the blood stream slow, and even emigration of leucocytes was observed, though to a much less degree than near the centre of irritation. At length the inflammation gradually subsided from without inwards towards the part at which the caustic was applied, the emigration of leucocytes ceased, the blood stream was restored, and the vessels returned to normal. Cohnheim got similar results with the cautery, and with other caustics applied to the frog's web.

From the above experiments we may draw the inference that active hyperæmia is not an essential part of inflammation, because (*a*) all the phenomena of active hyperæmia may occur without further results; (*b*) the active hyperæmia subsides before the emigration of leucocytes begins; (*c*) all the phenomena of inflammation can be sometimes observed without any hyperæmia. The essential phenomena of inflammation in a vascular tissue are therefore "*damage to the blood vessels*, with *stasis* and *leucocyte emigration*" (Greenfield).

II.—PHENOMENA OF INFLAMMATION IN EXTRAVASACULAR TISSUES.

The best position to study this set of changes is in the cornea of the

frog or rabbit. On the application of a caustic to the central part of the cornea, the vessels at the margin become enlarged and turgid, and the lymph channels and spaces become filled with leucocytes which have emigrated from the engorged vessels at the periphery. Under the influence of the irritation the corneal corpuscles become irregular in shape, and their processes become shorter and thicker. Amœboid movements of these protoplasmic masses are sometimes seen. Finally their nuclei divide, and the mass breaks up into several portions which may be regarded as new cells formed by proliferation. As a result of these processes the corneal tissue becomes opaque, softens, and may break down.

Therefore, as the vascular reaction is non-essential at the centre of the cornea during the inflammation, the chief phenomenon to be noted in inflammation of an extravascular part is the emigration of leucocytes.

EMIGRATION OF LEUCOCYTES.

Normally the white corpuscles chiefly occupy the peripheral stream in the blood vessels; but during inflammation this condition becomes more marked. At the beginning of stasis they are seen to accumulate in masses at the sides of the vessels. If watched for a time, the leucocytes are observed to send processes through the vessel walls, and to become altered in shape, so that they easily effect their exit, becoming extravascular, when they resume their normal shape. They are sometimes seen to go in the opposite direction to the blood current, so that their amœboid power must be greatly increased, which may be due to the great quantity of oxygen in the blood, as well as to the increased temperature of the part. There being a local dilatation of a blood vessel during inflammation, the intravascular pressure must be necessarily increased (according to a physical law), while, the external tissues being rendered more lax by the influence of the irritant, the extravascular pressure is decreased. These factors alone would increase the emigration of leucocytes, but if it be added that there is damage to the vessels themselves, one can easily understand why diapedesis should take place.

FUNCTION OF LEUCOCYTES IN INFLAMMATION.

(1) When leucocytes have escaped from the vessels, they make their way into the lymph spaces; in the case of membranes, to the surface. In doing so, they aid in the absorption of damaged parts, using deleterious and dead matter as food. When bacteria are present, the leucocytes may be seen attacking, and endeavouring to absorb them. Leucocytes which destroy bacteria in this way are called *phagocytes*. But on the other hand the bacterium is frequently poisonous to the cell, and often succeeds in killing it. (2) Leucocytes aid in the coagulation of lymph, as seen on the surface of a wound.

After inflammation there is found a greater number of leucocytes in the lymphatics, so it is most probable the great majority return into the lymphatics and blood vessels again, while some go to form pus corpuscles when suppuration occurs.

GENERAL RESULTS OF INFLAMMATION.

Inflammation tends to spread rapidly to surrounding tissues, which may be due to (a) simple extension of the change along the vessel walls; (b) irritation set up by the action of the products of inflammation; (c) septic material introduced from without.

Sometimes inflammation is attended by actual destruction of the tissue, if this takes place on a superficial part, we get what is known as "**ulceration**."

Inflammatory exudation differs from that of simple dropsy in containing a greater quantity of albumen, in having a higher specific gravity, and in being more coagulable. But the exudation from inflamed mucous surfaces, contains, as a rule, no fibrin, and does not coagulate. This is probably due to the epithelial layer acting as a filter, and intercepting some of the constituents of the fluid exudation, for, on removal of the epithelium a coagulable exudation may be formed, as is exemplified by the action of heat or other irritants on a mucous surface.

SUPPURATION.

The process of inflammation may go on to what is known as "suppuration," the formation of pus corpuscles and serum. Pus is never found except as the result of inflammation, but is not a constant product. In children, or those of the tubercular or scrofulous diathesis, inflammation tends more readily to go on to suppuration, especially if the injury be external, when the air, or noxæ contained in the air, are the more easily admitted. Hence the supposition that a specific poison is necessary to produce suppuration. But suppuration may occur even when the air is excluded, as in purulent pleurisy. In such conditions, when suppuration does occur in an internal organ, it may be argued that septic germs had been conveyed to the part by the blood; but this has scarcely been proved. Well marked cases of suppuration have occurred, and yet on close examination of the affected tissues no micro-organisms were detected.

Pus is formed partly from necrosed tissue, and partly from exudation products of inflammation. It is a pale yellow coloured fluid of the consistence of cream, with a mawkish taste and a faint sickly odour. The reaction is neutral or slightly alkaline, and it has a specific gravity varying from 1030 to 1033. It does not spontaneously coagulate, but on the application of heat a coagulum is formed. The main elements are serum and pus corpuscles. The *serum* contains most of the proteids found in the blood, as well as cholesterin, fatty acids, peptones (occasionally), and if it has remained long in the tissues, various gases. The *pus-corpuscles* constitute from 17 to 20 per cent. of the whole. Many of them resemble

leucocytes, and in the fresh state may show amœboid movement especially on being gently warmed. With these corpuscles are seen *fat* droplets, and large masses which appear to be due to the aggregation of degenerated cells. The *salts* of pus consist of sodium chloride (in large proportion), earthy and alkaline phosphates, carbonates, and a little iron.

Origin of Pus-Corpuscles.—It is still a disputed point whether pus-corpuscles are produced from leucocytes alone, or from connective tissue corpuscles as well. That many of them are produced from leucocytes there is little doubt, although in chronic suppuration there is an increased number of white corpuscles found in the blood. But in inflammatory conditions apart from suppuration (*e.g.*, rheumatic fever), there is also found an increased number of leucocytes in the blood. Probably the process of inflammation causes increased activity in their formation.

Abscess.—When a circumscribed collection of pus occurs in the interior of the body, an abscess is the result. If the abscess be acute there is no defined wall, and its contents are partly pus, and partly necrosed tissue. If chronic, the abscess has a well defined wall, formed by an overgrowth of connective tissue. The contents are pure pus, and if the abscess be an old standing one there may be flakes of lymph.

Abscesses tend to burrow and form laige sinuses, and in their development dreak down healthy tissue.

TERMS APPLIED TO INFLAMMATION, DEPENDING ON THE STRUCTURE AFFECTED.

(1) "**Parenchymatous**" consists of inflammation of the essential tissues of an organ, as for instance the epithelium of the kidney, or the air vesicles in the lung.

(2) "**Interstitial.**"—This condition affects the connective tissue framework of an organ, and is usually chronic.

(3) "**Catarrhal**" is inflammation of a mucous membrane in which the epithelial structures participate. The blood vessels become greatly engorged; the epithelial cells on the surface swell, multiply, and are cast off, while the deeper layer proliferates and replaces the surface ones when the inflammation disappears. The mucous glands rapidly secrete an abnormal quantity of mucus, and if the catarrh is chronic the outlet of the mucous duct may become blocked, and give rise to the formation of a small cyst. At later stages the basement membrane becomes greatly swollen, and round the vessels the tissues are infiltrated with exuded lymph and emigrated leucocytes.

PROCESSES OF REPAIR FOLLOWING UPON INFLAMMATION.

The special factors in repair following inflammation, consist in the processes of growth by which new tissue elements are formed. These processes are not restricted to the sequelæ of inflammation, for they normally take place as physiological phenomena to repair tissue waste,—but following upon inflammation they are greatly exaggerated, which, in part, may be due to the increased nutriment derived from the exudation and emigration of leucocytes from the damaged vessels.

Different tissues vary much in their capacity for repair, some being never perfectly restored if much injured. Specialized tissues have a low capacity for repair. Ganglionic nerve cells have little or no power of repair; nerve fibres and muscular fibres are restored imperfectly; epithelium and glandular structures recover from the effects of slight but not of any severe injury; connective tissue on the other hand, repairs with comparative ease, and may even grow beyond the bounds of mere repair.

It is an established law that new epithelium is always produced from pre-existing epithelium; but in regard to the production of connective tissue, which has a more important bearing on the process of repair, there is diversity of opinion as to its methods of origin. According to Ziegler, connective tissue is formed from emigrated leucocytes; but so far there is no sufficient proof that such is the case. It is more likely that as in the case of epithelium, connective tissue is produced from pre-existing connective tissue, and the results of observations support this view.

The repair of connective tissue appears to be closely associated with exudation and cell emigration from blood vessels. The condition may, for explanatory purposes, be very well studied in pleurisy or pericarditis, where adhesions of the visceral and parietal layers have taken place. There occurs first a coagulable exudation, which appears to act as a temporary structure. This fibrinous deposit was at one time believed to be transformed into fibrous tissue, but what takes place is rather a substitution, for this unorganised material, of a true tissue. On the absorption of the exudation (which is effected by the leucocytes), the two layers of the pleura come in contact; and a proliferation of the connective tissue elements of the part having taken place on each surface of the membrane, forming the so called "granulation surface," these surfaces coalesce more or less completely, and bind the two layers of the membrane together. This organizing structure is nourished by minute blood vessels, which arise as off-shoots from larger neighbouring vessels, and are entirely embryonic in structure. These elementary blood vessels and connective tissue elements constitute "*granulation tissue.*" At first the vessels are surrounded by connective tissue corpuscles, which, nourished by the vessels, are finally transformed into connective tissue fibres.

Development of the vessels.—They begin by a process of budding from an ordinary vessel wall. There first occurs the swelling of a protoplasmic mass at the side of the vessel, and this enlarges and grows outwards as a bud with one or more nuclei. At the point where the bud

springs there is a depression formed inside the vessel. As this bud increases in size, division of its nuclei takes place, and ultimately it is transformed into a capillary tube. This small capillary elongates and anastomoses with similar vessels until a network of new capillaries is formed.

These capillaries may develop by other processes, *e.g.*, the ordinary physiological process of vacuolation when the blood corpuscles apparently develop spontaneously as in the embryo. These vessels always have a very elementary structure, their walls being composed of endothelial cells with a number of fibrous tissue elements around, so that they are very permeable. They have not the relation to arteries and veins that exists in ordinary capillaries, and whenever their function is performed they gradually degenerate and soon disappear.

REPAIR OF WOUNDS.

I.—HEALING BY "FIRST INTENTION."

This process of repair only takes place in perfectly clean wounds, for instance, in a cut with a sharp, clean knife. If the divided surfaces are at once brought into apposition, the actual injury inflicted is really very slight, and the process of repair is so rapid that there is little or no inflammation present. If a wound such as the above be examined immediately after the injury, the following changes are seen :—The edges are somewhat apart owing to the elasticity of the tissues, and there is effusion of blood. Soon the hæmorrhage subsides, owing to the contraction and retraction of the arteries and collapse of the veins ; and the capillaries become plugged up by the clotted blood. The effused blood clots on the surface of the wound, and so unites the two edges. If the successive changes in the wound be divided into stages of twelve hours each ; we find at the *1st stage* (12 hours after the injury), that the edges of the wound are swollen, and united by coagulable lymph resembling ordinary fibrinous exudation, the blood which was formerly there having been absorbed. The fibrin at this stage contains only a few leucocytes, and at the deeper part of the wound there may be a little blood from secondary hæmorrhage. There is no distention of the surrounding vessels, and the tissues are but slightly altered. *After 24 hours*, the fibrinous coagulum still unites the edges, but it shows marked fenestration and lamination like a diphtheritic membrane. A great emigration of leucocytes has taken place into the coagulum, and they are especially seen at the base and edges of the wound, where they even conceal the fibrin and appear to loosen it. The leucocytes are very active, and many show division of their nuclei. The vessels near the wound are engorged, but at this period there is no visible change in the connective tissue corpuscles. *After 36 hours*, the engorgement of the vessels is more marked than in the last stage, and in the vicinity of each original vessel a large number of newly developed capillaries form a plexus. These plexuses are best seen immediately beneath the wound, where a few of the embryonic vessels may be observed to penetrate into the fibrin. Side by side with the newly formed vessels, connective tissue corpuscles make their appearance from multiplication of the pre-existing corpuscular elements. The fibrin is beginning to soften, due to the invasion by the capillaries, which are gradually increasing in number and size. *After 48 hours*, there is no marked alteration as regards the margins of the wound. The dilatation of the vessels has subsided, and those which were cut across have become completely closed. Distinct connective tissue fibres are seen at the deep part of the wound, and the lymph has become completely vascularized. *After 72 hours*, the chief phenomenon observed is a rapidly progressing transformation of the connective tissue corpuscles into fibrous tissue. If the wound be examined on the *5th day*, we find the network of capillaries is beginning to disappear, but a few vessels are still seen extending towards the surface, surrounded by connective tissue fibres and corpuscles. All the original lymph is now completely absorbed, it being substituted by connective tissue and capillaries, forming the so-called cicatricial tissue. The epidermis has begun to grow from the margins of the wound, by multiplication of the epidermic cells. At the *7th day*, the fibrous tissue is found to be more dense and the individual fibres more distinct. The edges of the wound are now quite close together, leaving no interspace. The vessels are fewer in number, and the whole part seems to have resumed its normal appearance; except that there are no hair follicles, nor sebaceous glands, and no sweat ducts.

II.—HEALING BY GRANULATION, OR "SECOND INTENTION."

This method of repair occurs in all open wounds. The process is essentially the same as in healing by "First Intention," the main distinctive feature being that the "granulation tissue" is formed on a free surface. When an open wound is produced, as in the case of an ulcer, hæmorrhage necessarily occurs, and the blood clots on the surface. This clotted blood is removed, and replaced by colourless lymph. Later there appear sprouting through this lymph small red projections, which are first seen at the margins, but latterly extend over all the surface. These projections are the capillary buds forced out from the arteries. If the excavation be deep, with a great loss of substance, these granulations may fill up the cavity. At the margins of the wound a glaze appears over the granulation buds, and extends over the whole surface; it is

due to the semi-coagulable condition of the lymph. As in the simple incised wound, a great number of leucocytes escape from the engorged vessels, but in this case they get discharged from the surface, many of them being transformed into pus corpuscles. In this way the wound assumes a suppurating type. Running alongside of the small granulation capillaries, are rows of connective tissue corpuscles. At a later stage these develop into connective tissue, which tends to contract as the healing process approaches completion. The small capillaries diminish in number owing to the process of "endarteritis obliterans" taking place in the vessels from which they spring. At length the epithelium gradually grows over from the edges of the wound, and eventually unites in the centre, thus covering the whole surface. The ultimate result of the above changes is the formation of a fibrous scar, devoid of sebaceous and sudoriferous glands, as well as hair follicles.

REPAIR OF FRACTURES.

I. SIMPLE FRACTURE of a long bone. When the fracture occurs the periosteum is usually torn, and the surface of the bone may be laid bare for a considerable distance. The soft tissues are lacerated, and the blood vessels ruptured ; so that profuse hæmorrhage takes place round the fractured ends, the blood forming a coagulum. A few hours after the injury an inflammatory reaction sets in, which usually lasts from two to four days, but may not disappear until the sixth day. Direct union rarely occurs, probably it does so only in a fissured fracture. The broken ends are always united by "callus," which acts as a framework for the production of new bone, and is only a provisional structure The callus resembles the cancellous tissue in the interior of long bones, and is produced chiefly from the deeper layers of the periosteum, to a less degree from the bone marrow. The callus is divided into three layers ; (1) external or ensheathing, outside the bone ; (2) intermediate, between the ends of the bones ; (3) internal or medullary, inside the bone. When the bone is kept at perfect rest the amount of callus formed is very small and mainly of the intermediate kind. But, if the bone has not been rigidly fixed, callus is thrown out in large quantity, the external variety preponderating. When union is complete, the external and internal callus disappear except where they fill up gaps, the intermediate callus forming the permanent bond of union.

Microscopic Changes.—If the organ be examined after the inflammatory reaction has set in, we find active hyperæmia of the vessels, and infiltration of lymph and leucocytes into the tissues, giving the parts a swollen appearance. On the second day the inflammation begins to subside, and the deep layers of the periosteum show signs of cell division. Changes begin to appear in the blood vessels which were torn ; their endothelium undergoes proliferation. By the third or fourth day we have a layer of highly vascular germinating granulation tissue formed, in which can be observed various forms of cells, contained in a delicate fibrillated matrix. The superficial part of the periosteum now shows swelling of the fibres and proliferation of the cells, so that the whole membrane becomes much thickened. At this stage there occurs a process of differentiation. Small spicules of bone are formed near the original bone ; these increase in size, become united, and form a structure resembling loose cancellous tissue. The tissue lying between the spicules becomes transformed into a marrow-like substance, consisting of small cells, large nucleated cells, and fat cells, lying among a number of small blood vessels. The consecutive stages are the same as those in normal bone formation. Beneath the periosteum and near the fractured ends hyaline cartilage begins to make its appearance. The cartilage cells are formed by the granulation tissue cells enlarging and becoming encapsuled by a hyaline-like substance. These cartilage cells swell up, lose their capsules, and become transformed into bone corpuscles (osteoblasts), and the cartilaginous matrix undergoes calcification.

The formation of bone is also accompanied by its absorption ; though the cells which remove the bone are not large osteoclasts as in normal bone formation, but small cells. At a later period the callus is largely removed, any projections rounded off, and the bone modelled more or less to its former shape. But if the limb be kept in motion during the process of repair, other changes take place. The fracture may be united by fibrous tissue, forming a false joint ; the two surfaces become polished, a bursa forms, and it is even asserted that fibro-cartilage may grow over the ends of the disunited fracture.

The time necessary for the repair of a fracture varies according to the bone which is broken. It may be said that the phalanges require a fortnight ; the bones of the arm, five weeks ; those of the leg, seven weeks ; and the femur, ten weeks.

II. COMPOUND FRACTURE.—The process of repair in this case differs from that in a simple fracture by the formation of *granulation tissue* and usually a degree of suppuration setting in, due to the introduction of septic matter. The formation of bone takes place very slowly, occurring first in the deeper parts.

REGENERATION OF INJURED TISSUES.

CARTILAGE.—When cartilage is cut through it is usually replaced by perichondrial connective tissue, but should the gap be great, as in ulceration, union takes place by fibrous tissue. When the bone is injured, cartilage can be formed from periosteum.

ADIPOSE TISSUE.—After an injury, fatty tissue undergoes degeneration. The nuclei of the fat cells swell, and the oil gets absorbed, its position being occupied by a mucous fluid. The connective tissue of the part proliferates, and becomes transformed into adipose tissue by the deposition of fat in the interior of the cells.

MUSCULAR FIBRE.—Regeneration of striped muscle takes place from pre-existing muscle-cells. The so-called muscle-corpuscles grow into large cells, lose their contractile substance, and multiply by division of their nuclei. They become elongated and spindle-shaped, and then become converted into striated fibres. That other cells have the power of generating striated muscle is doubtful, and authorities differ on the point. Non-striped muscle may be either produced from its kind, or from connective tissue cells.

NERVOUS TISSUE.—The central nervous tissue or ganglionic cells are not reproduced after being destroyed. Nerves have a slight reparative power. When a nerve is divided, the part cut off from its trophic centre undergoes degeneration. Inflammatory changes are set up in the nerve-sheath, causing emigration of leucocytes between the nerve filaments. The white substance of Sewhann becomes swollen, opaque, and the nuclei divide. In two days the myeline coagulates and becomes granular, and at length breaks up into cylindrical masses, when it is removed by the emigrated leucocytes. The protoplasm swells, and the axis cylinder disappears, leaving in the nerve tube a number of enlarged and divided nuclei. Regeneration takes place from the central end of the divided nerve. A bulbous swelling is observed ; from this issue new nerve-tubes, some with merely axis cylinders, while others have a distinct medullary sheath. These nerve-tubes pass out and penetrate the old medullary sheath of the peripheral division, and unite with the old nerve. The divided ends of a nerve may unite though they are separated by some distance, but the process is very slow, and may take two or three months to be completed.

CHANGES IN VESSELS WHEN CUT OR LIGATURED.

The changes are similar in arteries and veins, and whether the vessel has been ligatured, or closed spontaneously after section. When an artery is divided there occurs a certain amount of contraction and retraction, due to the elasticity of the muscular coat, which to some degree lessens the lumen of the vessel. After some hæmorrhage takes place, the blood clots at the distal and central ends of the divided artery. At the central end the blood-clot is slowly produced, on account of the blood pressure behind it, so it is firm and conical in shape, and usually extends up the vessel as far as the nearest branch. The distal clot is small and soft, because the circulation is soon restored by the establishment of a collateral anastomosis. Secondary hæmorrhage is most apt to occur from the distal end, as an eddy stream is very liable to take place from the anastomosing vessels, and the clot being of a loose nature, tends further to allow the blood to pass out. The blood-clot which fills the lumen of the vessel produces a local irritation in the inner coat of the artery, resulting in multiplication of its tissue elements. The endothelium lining the vessel becomes swollen and multiplies by cell division. The clot is surrounded by these endothelial plates, and processes of fibrous tissue begin to penetrate into the clot. A large bud now makes its appearance, penetrates into the mass, of blood-clot, and increases in size, partly by absorption of the mass and partly by nourishment derived from an off-shoot of the vasa vasorum. This bud consists of a large granular protoplasmic mass containing three or four nuclei, and is produced from the endothelium of the inner coat. As the bud penetrates through the mass of coagulum it is accompanied by its vessel, and may meet a similar bud from the other side of the artery. The capillaries of each bud then unite, and soon vascularize the blood-clot, which is organized into granulation tissue. Ultimately this is replaced by connective tissue which shrinks, and the artery becomes a mere fibrous cord.

HÆMORRHAGE.

Hæmorrhage consists in an escape of blood, (with all its elements,) out of the vessels into a tissue, or upon a free surface. The escape of blood from the vessels may arise from rupture of the walls—"per rhexin." It may take place by an ulcerative process from without—"per diabrosin." Or, as in inflammation, it may occur by leakage—"per diapedesin." Spontaneous hæmorrhage may be due to (1) Disease of the coats, *e.g.*, aneurism ; (2) Impaired nutrition of the blood, as in fevers, poisonings, pernicious anæmia, &c. ; (3) Certain diseases in which the cause is more or less obscure — purpura hæmorrhagica, malignant jaundice, and hæmophilia. When the quantity of blood is very small, and forms more or less sharply defined red or brown spots in the tissues, they are called *petechiæ ;* if rather larger, *ecchymoses.* If hæmorrhage takes place from many vessels of an organ it is called *parenchymatous.* Solid tissue soaked through with blood is said to undergo *hæmorrhagic infiltration* ; when this infiltration is clearly defined and limited to a part of the organ it is called *infarction.* Hæmorrhage from certain positions receives names accordingly : from the nose—*epistaxis* ; from the stomach—*hæmatemesis ;* from the lungs, *hæmoptysis ;* from the urinary tract—*hæmaturia.*

Hæmophilia is a condition in which there is a remarkable tendency to bleed from very slight causes, and even when there is no evident cause. The hæmorrhage, also, is frequently uncontrollable. This

tendency to hæmorrhage is far more common in the male sex than in the female. Careful examination of the vessels has shown no abnormality of structure, and the blood appears to be natural. This remarkable condition appears to be hereditary.

Hæmatoma.—When a large mass of blood is effused into a tissue it coagulates; should it not be absorbed for a considerable time, it is called *hæmatoma* or blood tumour. Such masses are frequently found on the ears of lunatics, and their origin has some interest from its medico-legal bearing.

HYPERTROPHY.

Hypertrophy consists in the increase in size of an organ or tissue from overgrowth of its special structures, while it still retains its form and functions. Strictly, hypertrophy means an increase in size of the tissue element; while the term *hyperplasia* signifies an increase in number of the tissue elements. Hyperplasia is a part of the process of inflammation, and is always associated with hypertrophy. As there are a number of hypertrophies which occur independent of disease or injurious influence, they may be termed **physiological.** Such are seen in the enlargement of the uterus and mammæ in the course of pregnancy. Enlargement of the prostate in old men, though frequently the cause of disease, cannot be referred to any morbid influence, and may also be classed in this group.

Pathological hypertrophy may result under the following conditions:—(1) Increased nutrition to an organ. John Hunter transplanted the spur of a cock on to its comb (a highly vascular organ), and the spur grew large and very rapidly. In section of vaso-motor nerves hypertrophy may result from an increased blood supply to the part. It is stated that in some cases of section of the cervical sympathetic in young animals great hypertrophy of the skin over the head and face results. (2) Increased functional demand. Cardiac hypertrophy illustrates this well, whether it be due to stenosis of an orifice, or Bright's disease. In Bright's disease the lumen of the small vessels in the kidney is lessened, and because of deficient excretion of urea the blood degenerates, and hence the walls of the vessels throughout the body are impaired, so that greater force is demanded to propel the blood. As a result, the cardiac wall becomes thicker. In stricture of the urethra, when there is impediment to the outflow of urine, the bladder wall increases in thickness, and acquires great distensible power. When there is an obstruction at the pylorus, the stomach enlarges and its muscular coat undergoes hypertrophy. (3) Compensatory hypertrophy. If one kidney is damaged, its fellow takes up its work, and consequently increases in size. When one lung becomes collapsed or cirrhotic, the other lung enlarges and partly compensates for the condition. When the spleen is extirpated, it is asserted that the lymphatic glands take on its function and enlarge. (4) Pressure or friction obviously causes hypertrophy, providing the pressure be not constant. Intermittent pressure produces hypertrophy; constant pressure produces atrophy (Paget). A good example of this is met with when a tight boot causes excessive growth of the epidermis of the small toe.

TUMOURS OR MORBID GROWTHS (Neoplasia).

The term tumour used to be applied in a loose way to all swellings and enlargements, hence an aneurism or a hydrocele was considered a tumour. For clinical purposes this loose nomenclature is still employed. Pathologically a tumour may be defined as a new growth of tissue which is not reparative, and which exists independently of the requirements of the organism. Tumours have certain **characteristics.** When once formed they continue to grow, even although the cause be removed. Their shape is not controlled by any special law of growth; it is irregular, depending upon the resistance met with and the facilities for extending in any particular direction. The tissue of tumours, though very often of the same type as the tissue from which they spring, is seldom identical. A tumour may consist of one tissue or more. They nearly all contain a circulatory system, the vessels forming a complete network, and for the most part are newly formed. They have always a framework of connective tissue, forming a supporting structure, but which is not a proper constituent of the tumour. Most of them contain lymphatics, which communicate with the normal lymphatic channels. Many of them contain nerves, and are consequently sensitive. A tumour that resembles in structure the tissue in which it grows is called *homologous*; if it differs in structure, it is termed *heterologous*.

AETIOLOGY OF TUMOURS.

Although the cause of morbid growths is frequently obscure, yet there are various conditions which predispose to their occurrence. That the epithelioma, for instance, rarely occurs before advanced life, shows that an impaired vitality of the tissues favours the growth. Tumour growth is influenced by the following causes:—(1) Heredity. That heredity is a frequent cause is abundantly proved by clinical observation. (2) Continued irritation. The epithelioma of the tongue or lip usually results from the smoking of a short pipe, or the irritation set up by a decayed tooth. Chimney-sweeps' cancer (an epithelioma of the scrotum) is caused by the

mechanical irritation produced by contact with the soot. Cancer of the stomach is most common at the pyloric end, and is supposed to be due to the continued friction caused by the passage of food into the intestine. A malignant tumour in the intestinal canal most commonly arises at the lower part of the bowel, and is probably the result of the decomposition of fœcal matter. Cancer of the bladder is frequently a sequel to a calculus. (3) Variations in nutritive activity. This is probably the main cause of tumour growths occurring in organs which have a cyclical function, such as the ovaries, uterus, mammæ, &c. (4) "Embryonic residues." This is a theory advanced by Cohnheim to account for the production of certain tumours. He holds that in an early stage of embryonic development more cells are produced for the formation of any particular part of the body than are required for the purpose, so that a number of embryonic cells remain unemployed. Such a residue may remain indefinitely quiescent, but on receiving some stimulus, which may be in the form of increased nutrition, it starts independent growth, and gives rise to a tumour. Tumours which occur where two different layers of embryonic tissue come in contact are cited as examples of this class, such as in the position of the branchial clefts, vagina, &c. It is sometimes found that in case of "rickets" a cartilaginous tumour is developed on the hand. This may be due to an embryonic residue of cartilage which in the course of the disease receives increased nutrition, and is thus stimulated to renewed growth.

When once formed a tumour tends to increase in size. Its growth may be entirely local, or it may affect distant parts of the body. Locally, it increases in size by infiltration of the surrounding normal tissues. It may absorb these tissues as pabulum, or transform them into its own elements. Generally, a tumour spreads by its blood vessels or lymphatics, through which cell elements are conveyed to a distant tissue, and these multiply there.

Morbid growths frequently undergo degenerative changes. Among the more common is fatty degeneration, which may be followed by caseation and calcification. Tumours may also change from one type of tissue to another (metaplasia). The rapidity with which tumours grow varies much. A tumour the size of a marble has been known to exist on the face of a man for twenty-five years, and then take a fresh start and grow to the size of an orange. The constitutional effects of a tumour may be nil; while, on the other hand, they may be very severe, giving rise to cachexia. Cachexia only results from malignant growths, especially if these growths ulcerate and emit a discharge.

CLASSIFICATION OF TUMOURS.

Histological.—Tumours have been classified according to the tissue of which they consist. Those of fibrous tissue—whether embryonic or fully grown—would therefore be included in one group. Thus, an ordinary fibroma and a sarcoma would come under the same division.

Embryological.—Some propose to classify tumours as mesoblastic, epiblastic, and hypoblastic, according to the embryonic origin of the tissue of which they consist.

Clinical.—For all practical purposes this classification is by far the best. It may be considered a combination of the histological and embryological classifications. (1) **Simple tumours** are composed of tissue elements which are found in the healthy body; they are mesoblastic in origin, and therefore chiefly consist of connective tissue. These tumours grow slowly, are usually single, they give rise to no inconvenience except by their mechanical effects, no secondary growths arise, and if removed they do not recur. They usually have a capsule of fibrous tissue, which is derived from the surrounding connective tissue by a process of chronic inflammation. These general characters lead us to speak of such tumours as "innocent" or "non-malignant." (2) **Sarcomatous tumours** are composed mainly of embryonic connective tissue, with sometimes an attempt at the development of a higher form. They are all derived from the mesoblast; they are all flesh-like in consistence, and under the microscope resemble granulation tissue. In point of malignancy they occupy a position midway between the simple and the cancerous forms. (3) **Cancerous tumours** consist of overgrowths of epithelium, with a connective tissue basis, and may be derived from mesoblast, hypoblast, or epiblast. These tumours are highly malignant. They grow rapidly, destroy the part in which they grow, produce cachexia or constitutional disturbance, they give rise to secondary growths in other parts of the body, and when removed they recur.

I. (A) SIMPLE TUMOURS.

MYXOMA.

The tissue of which this tumour is made up is the chief form of connective tissue in the fœtus; in the adult it is only found in the vitreous humour. They are usually small, more or less lobulated, jelly-like in consistence, and present the appearance of boiled tapioca. A capsule of fibrous tissue surrounds them. From this capsule delicate trabeculæ are sent into the growths, and form supports for the blood vessels.

Microscopically, large, nucleated, irregular, branching cells are seen lying in a jelly-like matrix which is composed of mucin. As the bulk of the tumour is usually made up of œdematous connective tissue, fibrils of this form of tissue are to be observed scattered irregularly through the matrix.

Sites of occurrence.—(1) Submucous tissue (nasal polypi). (2) Growing from the villi of the chorion (hydatid mole). (3) Connective tissue in various positions.

There are several varieties of this tumour, vizt. :—myxo-fibroma, myxo-chondroma and myxo-lipoma.

GLIOMA.

Gliomata are tumours which develop from the neuroglia of the central nervous system, and when fully grown are largely made up of neuroglia cells. They are met with in the brain, and more rarely in the spinal cord. They are grey in appearance, and imperfectly marked off from the surrounding healthy tissue. They are small tumours, growing slowly, and are distinguished from the small "round-celled sarcoma" by their firmness.

The **microscope** reveals a mass of neuroglia cells, the nuclei of which are distinctly seen. Some of these cells have two and three nuclei, and an intranuclear plexus is frequently observed. These protoplasmic masses send out numerous delicate ramifying processes which anastomose with each other. The vascularity of the tumour varies, but frequently the vessels are dilated at various points. Researches have shown that the cells are derived from the normal neuroglia, and the ganglionic cells take no part in the proliferous process. The so-called glioma of the retina is a form of round-celled sarcoma.

FIBROMA.

Fibromata are tumours made up of white fibrous tissue with a varying number of connective tissue corpuscles. They are sharply defined, dense, dry, and firm, and contain few blood vessels. They are rounded or lobulated, have a capsule, and on section present an appearance compared to watered silk. Fibromata may occur in any structure which contains fibrous tissue.

Classification I. **Fasciculated Fibroma** occurs most frequently in subcutaneous and submucous tissues ; in the upper and posterior part of the pharynx ; in fasciæ, and in the sheaths of nerves. They may be soft or hard in consistence. (a) *Hard fibroma* is usually round, firm, and tough, and on section presents a glistening appearance. It is made up of interlacing bundles of white fibres, with very few connective tissue corpuscles. (b) The *soft fibroma* is an earlier form of the hard, and besides being softer it grows more rapidly, and contains more vessels. The connective tissue fibres are widely separated, with a good deal of fluid between them, and their arrangement is feather like. The corpuscles are more numerous than in the hard variety. The more cellular these tumours are the more rapid is their growth.

II. **Lamellated** or flat fibroma arises from the serous covering of internal organs, and may be regarded as the result of a chronic inflammation of the serous surface. It consists of flattened bands of fibres arranged in lamellæ with very few connective tissue corpuscles between.

A "**Keloid**" is a small fibrous tumour which arises from the cicatrix of a cut or burn.

"**Molluscum fibrosum**" usually partakes of the nature of a soft fibroma, but contains more blood-vessels, and a great deal of fluid. It is always multiple, and frequently occurs on the abdominal wall.

LIPOMA.

Fatty tumours usually arise from normal adipose tissue. They are often multiple and of large size. They may be rounded or lobulated, and are frequently pedunculated. Fatty tumours are usually pale yellow in colour, and are softer and more plastic than ordinary fatty tissue.

Microscopically, they reveal the ordinary structure of fat, with here and there a few fibrous tissue fibrils binding the mass together. Frequently some myxomatous tissue is found mixed with the fat. Although fatty tumours may arise from any position where fat exists, their most common site is subcutaneous tissue, especially over the shoulders and buttocks. Lipomas have been found in tissues which do not normally contain fat, such as the dura mater, the submucous tissue of the intestine, and bone.

CHONDROMA.

Chondromata essentially consist of cartilage, which is usually hyaline, but always contains a certain amount of fibrous tissue. They are usually multiple, rounded or lobulated, and surrounded by a fibrous tissue capsule. As a rule they are firm, though in some cases they are found soft owing to mucoid degeneration. On section they cut with the peculiar creak of cartilage, and present a translucent appearance.

Microscopically.—The structure of hyaline cartilage is seen, but there is usually some form of degeneration to be observed also. The cartilage differs from normal hyaline cartilage in having fibrous septa containing blood vessels, and dividing the structure into lobules. The cells also frequently branch, and have no distinct capsule.

Chondromata chiefly occur in young people, when the bones are in a growing condition. Their most common sites are in the periosteum of bones; in the bones themselves; in the parotid and other salivary glands; in the testicle, skin, lung, and mammæ. When growing from the periosteum their most frequent positions are the ends of the metacarpal bones, and on the phalanges of the fingers and toes. When they

occur in structures normally devoid of cartilage, their probable origin is from small bits of cartilage that have been entangled in the tissue, and escaped ossification.

The **varieties** of this tumour are rather numerous. (1) The *myxo-chondroma* has its origin in bone. It is a transition between cartilage and mucous tissue, and consists of irregular branching cartilage cells lying in a mucoid matrix. They are a malignant form of growth. (2) *Chondro-adenoma.*— A rare form of tumour found in glands, and made up of chondromatous tissue with a certain amount of glandular structure. (3) When myxomatous degeneration appears in the chondro-adenoma we get the *myxo-chondro-adenoma.* (4) Sometimes a chondroma occurs combined with sarcomatous tissue—*chondro-sarcoma.*

The degenerations to which these tumours are most subject are mucoid softening of the matrix, calcification, and true bone formation.

OSTEOMA.

Bony tumours are usually outgrowths of bone, and most frequently arise at the junction of a bone and its cartilage. These tumours have been classified according to their position of occurrence and extent of growth. Those growing from the exterior of a bone are called *exostoses;* from the interior of a bone *enostoses.* Should the growth be extensive and diffused in a bone the term *hyperostosis* is employed ; when the new formed tissue is quite localized, and gives rise to very little thickening it is described as an *osteophyte.*

Classification.—I. **Eburnated** osteoma is a hard, polished, ivory-like outgrowth, growing from the inner table of the skull, and appears to be of syphilitic origin. They have no Haversian canals, and therefore no blood vessels ; but there are canaliculi similar to those found in the cement of teeth, which run towards the surface (Ranvier).

II. **Compact** osteoma is met with as a nodular growth beneath the periosteum, or in the periosteum of long bones ; but it may be found in other positions even apart from bone. The structure very much resembles ordinary compact bone, but the Haversian systems and blood vessels are arranged at right angles to those of the old bone, and are more irregular.

III. **Spongy** osteoma is a growth which arises commonly from epiphyseal lines. It is also met with in old people as a false exostosis, being really an ossification of the tendon of a muscle. The tumour is spongy in consistence, and the trabeculæ are much thinner and not so numerous as in the compact form.

The above tumours are designated *true osteomata,* in contradistinction to other forms of tumour growths which may ossify as a degeneration, *e.g.,* chondroma. Malignant tumours also sometimes form bone in their structure, *e.g.,* osteo-sarcoma. When excrescences occur in connection with the teeth, and consist of cement or crusta petrosa they are called "*dental osteomata*"; if they consist of dentine they are odontomata.

ANGIOMA.

Angiomata are tumours composed of blood vessels, and are divided into simple and cavernous.

I. The **simple angioma** is a tumour made up of blood vessels which are supported by some normal basis tissue. The vessels may be either arterial, venous, or capillary in character; most frequently they are venous and capillary, and are either newly formed, or derived from pre-existing vessels more or less altered. Although there may be fusiform dilatations met with on the vessels here and there, yet the general tubular form of the vessel is maintained. These tumours are chiefly met with as congenital growths in the skin (nævi), where they appear as bright red or livid patches, not raised from the surface.

II. The **cavernous angioma** is distinguished from the "simple" by the fact that the tubular form of the vessels is lost. This tumour is made up of large irregular spaces containing blood; the walls of these cavities are formed of fibrous septa lined with endothelium. In structure it resembles the corpus cavernosum of the penis. This form may be developed from the simple angioma by obliteration of the septa, two or more vessels going to form one large space. They most frequently occur in the liver substance near the surface, less frequently in other viscera. In the liver they are never congenital, but are developed in advanced age when the liver is tending towards atrophy.

III. **Hæmorrhoids** may be considered as angiomata formed in the mucous membrane of the rectum by a growth mainly of the submucous tissue, and of blood saccules derived from small veins by morbid dilatation.

LYMPHOMA.

A lymphomatous tumour strictly consists of the development of new lymphatic tissue; but the term lymphoma is used in a comprehensive sense, and includes formations which are not really tumours. Thus a lymphatic gland or follicle increases in size when there is a simple multiplication of its lymphoid cells and hyperplasia of the reticular tissue. These enlargements of lymphatic glands may be the result of inflammation, and are difficult to distinguish from a true lymphoma. The lymphoma is always found in positions where lymphoid tissue is normally present, and usually occurs as a solitary mass not attaining any great size. When rapidly growing they are generally soft and pinkish white on section; on scraping there exudes a juice containing epithelioid cells, a circumstance which gave rise to the supposition that the tumours were cancerous. They tend to be malignant, and infiltrate

the surrounding tissues. The more slowly growing forms become indurated. **Microscopically** these growths present the appearance of adenoid tissue; but the reticular stroma may be small or increased beyond the normal, and the endothelial plates are usually well developed.

The lymphoma is most frequently found in the mediastinum, kidney, spleen, and uterus.

Varieties.—I. **Lymphosarcoma.**—This variety of lymphoma is really a form of the small round-celled sarcoma. It most commonly originates in lymphatic glands, and may then be distinguished from the ordinary lymphoma by its very rapid growth. Its clinical history, appearance, and soft consistence make it liable to be mistaken for a cancerous tumour. The microscope shows the adenoid reticulum and endothelium to be scanty, while there is a great increase in the number of lymphoid cells.

II. **Lymphadenoma.**—This form of growth occurs in "Hodgkin's disease," and, as its name implies, chiefly consists in an overgrowth of the adenoid reticulum in lymphatic glands. The endothelial plates are also increased, but there is generally a diminution in the number of lymphoid cells. The lymphadenoma is usually indurated and fibrous, but when caseation sets in it becomes soft.

III. **Lymphangioma.**—This tumour essentially consists in the dilatation of a mass of lymphatic vessels associated with hypertrophy of the vessel wall. The dilatations may be very great, and actual cysts may be formed in them. These cavities contain a clear form of lymph. As in the case of the blood tumour, the lymphangioma may be congenital or acquired, simple or cavernous. The congenital form may be met with in the tongue (macroglossia), lips (macrochelia), skin (lymphatic nævus), labia, &c.

MYOMA.

There are two varieties of this tumour, those consisting of striped muscle (rhabdomyoma), and those composed of non-striped muscle (leiomyoma).

I. The **non-striped** variety occurs by far the most frequently in the uterus, though it may be found in any position where non-striped muscle exists. They appear as spherical, hard, nodulated growths, pale in colour, and not unlike fibromata; but they are more elastic, and have a more regular arrangement of their tissue. In the uterine wall the tumour may grow—*(a)* in the muscular wall itself (intramural); *(b)* beneath the mucous coat, projecting into the cavity of the uterus as a polypoid growth (submucous); *(c)* from the outer surface of the uterus, when they grow into the peritoneal cavity (subperitoneal). The muscular tumours of the uterus always contain some fibrous tissue as well as muscular, hence the term *uterine fibroid*. These tumours are composed of small bundles of spindle-shaped fibre-cells, interlacing in various directions. Between the bundles there is fibrous tissue binding them together. A section may reveal one of these bundles cut transversely, when nucleated and non-nucleated round cells appear. There are very few blood-vessels seen in this growth.

II. The **striped** muscular tumour is almost unknown apart from congenital growths found in children. They have occurred in the heart, kidney, and voluntary muscles. Forms of this tumour have been met with in the testis and kidney combined with sarcomatous tissue *(myosarcomata)*.

NEUROMA.

Neuromata are divided into two classes, according to whether they consist of true nerve tissue, or are mere hyperplasis of the supporting elements of such tissue.

I. The **true nerve tumour** is a growth of rare occurrence. It may be composed of nerve fibrils or ganglionic cells. When made up of nerve fibrils its most frequent site is at the end of divided nerves, as in amputation stumps ; and even then there is associated with the enlargement a great deal of fibrous tissue. Neuromata are very often multiple, never large in size, and their long axis frequently coincides with that of the nerve.

II. The **false neuroma** is in reality either a fibroma or myxoma occurring on the nerve trunks, or a glioma in the central nervous system. On examination, nerve fibres are seen to run through these structures ; but the nerve tissue is unchanged, and there is no evidence of new growth of the nerve fibres.

I. (B) INNOCENT EPITHELIAL TUMOURS—GROWTHS COMPOSED OF MORE THAN ONE TISSUE.

PAPILLOMA.

Papillomata occur most commonly on the skin, and include all those growths known as corns, warts, and horns, as well as the cauliflower excrescences so often found on the genital organs of gonorrhœal patients. They also arise from mucous membranes, especially in the larynx and alimentary tract. These tumours may grow rapidly, but are nevertheless non-malignant. The essential element in the development of a papilloma consists in an overgrowth of epithelium on a free surface, with hypertrophy of the subjacent tissue, the epithelium retaining its normal position, however, to the structures beneath it.

Microscopic appearance.—In a typical papilloma of the skin, the tumour is seen to have a well developed fibrous tissue basis, supporting a great number of blood-vessels, some of which are dilated into

sinuses. The ordinary papillæ are very much enlarged, branch in various directions, and contain a good deal of connective tissue with a few vessels. The epithelium covering this is present with all its layers, but the "rete Malpighii" and "stratum granulosum" are enormously developed, while the "horny layer" is very thin. If, however, the tumour be a horny papilloma, the "stratum corneum" forms a layer of considerable thickness over the stratum granulosum. The horny growths arise chiefly from the face and neck, where there is a secretion of a large quantity of sebaceous material. Here the horny layer, instead of being constantly shed, is glued together by this sebaceous excretion, which dries and forms a horny mass.

The distinctive features which characterize these tumours from an "epithelioma" are that the normal relation between the epithelium and subjacent tissue is maintained, the epithelial cells of the papilloma never invade the connective tissue as in epithelioma, and the papilloma does not infiltrate locally nor give rise to secondary growths.

ADENOMA.

The adenoma is a tumour composed of tissue resembling that found in secreting glands. It may consist in the formation of new glandular tissue, or in the hypertrophy of pre-existing gland structures. Adenomata are unable to produce the normal secretion of a gland. The adenoma is usually a small, firm tumour, with a fibrous capsule sharply marking it off from the surrounding tissue. When it occurs in the breast it simulates a scirrhous cancer, but there is never retraction of the nipple as in the latter growth.

Microscopically the mass is seen to consist of a well developed fibrous tissue matrix, supporting a number of blood-vessels; here and there are observed newly formed gland-tubes in various stages of evolution. Some of these tubes are well developed, others may be dilated into cysts. On closer examination the tubes are found to consist of a delicate layer of flattened cells (Debove's layer), lying on the fibrous basis. Above Debove's layer there is a single layer of columnar or cubical cells. Each cell has a distinct nucleus, but there are no cilia present. If a section of a tumour be made, passing obliquely through one of these glands, the appearance presented may lead one to suppose that the gland-tube was made up of more than two rows of cells because an elongated view of both layers is obtained.

Adenomata are found in the breast (commonest position), parotid gland, testicle, and mucous membranes of the air passages and alimentary canal.

Varieties. I. **Spurious Adenoma.**—This tumour is in reality not an adenoma, but merely consists of a hyperplasia of tissue between the ducts and acini of a gland. It is most frequently a fibroma or sarcoma.

If such a sarcomatous tumour be excised, the growth recurs, but is then devoid of glandular structure.

II. **Cystic adenoma** ("compound ovarian"). This compound cystic tumour grows in the ovary or broad ligament, and frequently reaches a very large size, often filling the abdominal cavity. It consists of a large number of cysts enclosed in a fibrous capsule, the cysts being separated from one another by trabeculæ sent in from this capsule. There is usually a large "mother cyst" occupying the centre of the mass. The large cysts contain a watery fluid, with sodium chloride and a very little albumen. The fluid is usually altered in colour by blood pigment. The small cysts contain a gelatinous material, with a greater quantity of albumen.

Microscopically.—Upon the highly vascular connective tissue stroma Debove's layer rests, as seen in the ordinary adenoma; above this is a layer of columnar epithelium, usually in the form of chalice cells, with their nuclei placed in the lower third of each cell. These chalice cells secrete mucus.

This tumour is simply an adenoma in which the tubes have been distended, by retained secretion, into cysts.

The *adenoma* and *papilloma* are closely allied to one another. In the ~~one~~ case the epithelium forms a covering for the connective tissue and blood-vessels, while in the other it forms a lining for these structures.

II. SARCOMAS.

SMALL ROUND-CELLED SARCOMA.

This tumour consists of the most elementary form of connective tissue. They are very malignant, grow rapidly, and may reach a large size. They are found as soft masses, pale pink in colour, and well defined from the surrounding healthy tissue, although they have no capsule. All that is seen **microscopically** is a large number of small round nucleated cells, with very little intercellular substance. Here and there may be observed an embryonic blood-vessel formed by spindle-shaped cells. These tumours occur chiefly in fasciæ, subcutaneous tissue, connective tissue of the nerve centres, retina, bones, testicle, and mammæ as primary growths, but do not affect the lymphatics. Secondary growths always occur first in the lungs, after which they may arise anywhere. Next to the "melanotic sarcoma," the small round-celled form is the most malignant of all sarcomata.

LARGE ROUND-CELLED SARCOMA.

This form of growth occurs in much the same positions as the small round-celled sarcoma, but most frequently arises from the sub-mucous tissue of the pharynx and posterior nares. It appears as a pale, firm,

small tumour, with a sharply defined outline. It is not nearly so malignant as the above described form of sarcoma, and rarely gives rise to secondary growths. In structure, this tumour is made up of the same tissue elements as were described in the small round-cell form, but the cells are very much larger, and many of them are multi-nucleated.

SMALL SPINDLE-CELLED SARCOMA.

Sarcomata consisting of spindle-shaped cells are among the most common of all tumours. They are much firmer in texture than the round-celled variety. Of the small spindle-celled sarcoma there are usually two forms described. (1) The "recurrent fibroid" (so called from its firmness and great tendency to recur locally), is a tumour which varies much in size. It is the least malignant of all sarcomata, seldom giving rise to secondary growths, and having no tendency to local infiltration. The cells are not typically spindle in outline, but rather oat-shaped. (2) This tumour very much resembles the recurrent fibroid, but is not so smooth in character. The cells are complete spindles, though many of them appear on section to be round nucleated masses of protoplasm, while others have no nucleus. This is due to some of the cells being cut transversely through their nucleus, while sections of many are made near the end of the cells. Because of this many of these tumours are mistaken for round-celled sarcomata by a casual observer.

These tumours contain embryonic blood-vessels resembling in structure those found in the round-celled sarcoma, but they are not quite so numerous. They may be found growing from almost any position where there is connective tissue, but their most frequent origin is from the periosteum and inner surface of the dura mater.

MYELOID SARCOMA.

The myeloid or "giant-celled sarcoma" is a tumour composed of small spindle cells, with a number of large irregular cells scattered throughout its substance. They are large tumours of slow growth, occurring either in the interior of a bone or below its periosteum. When they grow from the interior of a bone they expand until the bony structure around them merely consists of a thin shell. These tumours are found as firm elastic masses, usually pale in appearance, but frequently discoloured from hæmorrhage. In the interior of the mass fragments of bone may be found, and the presence of small cysts is not uncommon. **Microscopically,** they are seen to consist of small spindle cells, with here and there a large irregular multi-nucleated mass of protoplasm—the so-called "giant cells." In structure these giant cells resemble the osteoclasts found in growing bone, but are usually very much larger, containing sometimes as many as twenty or thirty nuclei.

The position in which the tumours grow suggests the origin of these large cells, and this might serve to illustrate the general law that tumours reproduce the physiological type of tissue from which they spring. These growths most frequently arise from the lower end of the femur, the upper end of the tibia and humerus, the lower jaw (malignant epulis), and the antrum of Highmore.

Although the above may be considered the typical form of this variety of sarcoma, yet giant cells may be found in any sarcoma growing in connection with bone, and are frequently associated with the large spindle-celled sarcoma.

LARGE SPINDLE-CELLED SARCOMA.

This form of growth is softer, of a deeper colour, and more vascular than the small spindle-celled sarcoma. They do not usually reach such a large size, but may grow rapidly, and are upon the whole of a much more malignant type than the last named form of tumour.

The cells are very much larger than those of the small-celled sarcoma, but still they have very little intercellular substance. In shape they are irregular, and frequently dove-tail into one another by their bifurcated ends. The embryonic blood-vessels are very numerous, and the picro-carmine stain often reveals green masses which consist of extravasated blood.

The chief position in which this tumour is found is the skin, and from this the lymphatic glands are often affected.

MELANOTIC SARCOMA

Is a growth which cannot be mistaken for any other form of tumour, as its dark blue-black appearance is very characteristic. It does not usually grow to a large size, and as a rule arises from those positions which normally contain pigment—choroid, retina, pia mater, and skin. **Microscopically**, the growth is seen to be made up of large spindle cells, though sometimes there is a melanotic form of growth composed of large round cells. Around the nuclei of many cells is found a dark brown pigment—melanin—which appears to be elaborated by the cells. This pigment is to a less degree intercellular. Numerous vessels are present in the tumour, and owing to their embryonic structure hæmorrhages occur frequently, so that a collection of altered blood pigment may be observed round these vessels, which should not be mistaken for melanin.

Of all sarcomata the melanotic is the most malignant. Secondary growths begin to make their appearance when the tumour reaches the size of a marble. These secondary growths are usually pigmented, though sometimes they are altogether devoid of colouring matter.

PSAMMOMA.

Psammomata are growths which arise in certain definite positions, viz.,—the cerebro-spinal membranes, especially in the region of the

choroid plexus and pineal gland. In size they rarely exceed a small pea, and consequently from a clinical point of view are of little importance. They are usually globular in shape, and are composed of concentrically arranged spindle cells with a large number of blood vessels. The centre of the growth is occupied by masses of calcareous matter, which very much resembles the ordinary cerebral sand. The tumour may consist of fibrous or myxomatous tissue instead of sarcomatous; when so, it is non-malignant.

ALVEOLAR SARCOMA.

The alveolar sarcoma is a tumour which may be mistaken for a cancer, because of its arrangement of alveoli supported by fibrous tissue. If the following points be attended to the distinction will become apparent. (1) The cells are round and simple, not of an epithelial type; (2) there is usually some intercellular substance passing inwards from the fibrous septa, and partly enclosing each cell; (3) the tumour originates from connective tissue. As in the case of cancer the blood vessels are supported by the fibrous tissue stroma, and do not run into the cell groups.

These tumours arise as small growths, which are most frequently found on the true skin, and in the pia mater, muscle, and bone.

OSTEO-SARCOMA

Is a tumour which is always found as a primary growth in connection with bone. As regards their cell structure they vary much, but they have always a tendency to form bone both in their primary and secondary growths. The cells have an arrangement like normal spongy bone, and even Haversian canals may be distinguished.

OSTEOID-SARCOMA.

Unlike the previously described tumour, there is no attempt at true bone formation in the osteoid-sarcoma. It is a very malignant form of growth, arising primarily from periosteum, and affecting secondarily the lungs, serous membranes, etc. The cells are large and multinucleated; and the intercellular matter becomes infiltrated with a substance like that found in the matrix of cartilage, after which it is infiltrated with lime salts. Consequently the tumour presents the appearance of a hard mass, which is apt to be mistaken for a bony growth.

ENDOTHELIOMA

Is a term applied to psammomata, alveolar sarcomata, and other growths originating in, and composed of endothelium. "If the endothelium of the serous membranes lining the great cavities of the body be regarded, in accordance with modern embryological views, as derived from the hypoblastic epithelium, the distinction intended by the word endothelioma becomes important" (Payne).

DEGENERATIONS AND MODIFICATIONS OF STRUCTURE IN SARCOMATA.

Fatty, hæmorrhagic, hyaline, and myxomatous are the most common degenerations for these tumours to undergo.

They may become modified by the cells taking up oil droplets and giving rise to "lipomatous sarcoma." Pigmentary changes may arise to form a melanotic sarcoma. The intercellular substance may undergo mucoid degeneration, leading to the formation of a myxo-sarcoma, or become calcified or even ossified.

III. MALIGNANT EPITHELIAL TUMOURS (CANCERS).

A. EPITHELIOMA.

Epitheliomata are tumours which are conveniently classified apart from "true cancers," which will be next considered. They originate by an excessive overgrowth of epithelium, which invades the subjacent tissues and lymphatic system. They are very malignant tumours, and are specially liable to produce secondary growths in the lymphatic glands. They are classified according to the surface from which they arise.

I. SQUAMOUS EPITHELIOMA.

An ordinary papilloma, if subjected to irritation, may give rise to this form of tumour—the interpapillary epithelium of the epidermis growing down beyond its normal limits. But they usually arise primarily as malignant growths, especially at the junction of the skin and mucous membrane, which are parts not only exposed to great friction, but are also the seats of great proliferative activity. The lips, tongue, nose, anus, penis, and orifice of the vagina are the most common sites.

This tumour usually occurs as a warty-looking outgrowth, with an ulcerated centre and indurated stony margins. There is usually an exudation of ichorous fluid, and the nearest lymphatic glands are frequently indurated.

Microscopically, all the layers of the normal epithelium are observed to be well developed, especially the horny layer, and the germinal layer of the rete Malpighii. The epithelium grows down into the subjacent connective tissue by the lymph spaces; in doing so irritation is set up, which causes overgrowth of the connective tissue elements, and the part becomes more vascular. As columns of the epithelium thus infiltrate the subjacent tissue, the cells are gradually removed from the germinal layer and carried to the centre of the column, where (instead of being cast off, as they would be were it a free surface) they undergo colloid or horny changes, and form a hard centre,

against which succeeding layers of cells are projected and flattened. This gives rise to a rounded mass, showing a concentric lamination and homogeneous interior, constituting the so-called "cell-nest."

The number of cell-nests in such a tumour depends on the rapidity of the growth. Where the tumour grows slowly there will be not only more cell-nests, but those present will be more perfectly formed; while, if it be a rapid growth, there may be no such cell-nests present. Owing to the downward extension of the proliferating epithelium into the lymphatic spaces, epithelial cells are very apt to become detached, reach the nearest lymphatic gland, and so give rise to a secondary growth. The ulcerated centre, which tends to spread to the margins, is due to want of nutrition, the lymph being unable to percolate through the thickened layer of epidermis.

II. COLUMNAR-CELLED EPITHELIOMA.

(SYNONYMS—"MALIGNANT ADENOMA," "ADENOID CANCER.")

This tumour arises from surfaces covered by columnar epithelium. In this case the epithelium invades the subjacent tissue, and gives rise to spaces or tubes, which become lined by the columnar cells. The tubes are supported by a stroma of young connective tissue, which may be regarded as a growth due to the irritation set up by the invading epithelium. Sometimes the alveoli are lined by a double layer of columnar cells, but this more frequently happens in secondary growths. The alveolar cavities, though usually empty, may contain a few cell elements which have become detached from the fibrous stroma. For this reason it is sometimes difficult to distinguish this tumour from a true cancer. Should the cells in the alveoli multiply, its structure is then essentially that of a cancerous growth.

The columnar-celled epithelioma varies in appearance and consistence according to the position in which it is found. In the stomach it is met with as a flat mass, usually soft in consistence, and therefore may be mistaken for "colloid cancer." In the rectum it is pale in colour, soft in consistence, and frequently ulcerates. It is found in the liver usually as scattered nodulated masses, and in appearance looks very like true cancer of the scirrhous variety. Besides the above-mentioned positions, it frequently arises in the large intestine, in the gall bladder and bile ducts, and in the bronchi.

B. CARCINOMA (CANCER PROPER).

Cancers are tumours which grow from glandular epithelium. They have always a very definite stroma of fibrous tissue, which forms a series of spaces or alveoli. The alveoli contain cells of an epithelial type. These cells have usually a cell-wall, and contain one or more nuclei;

they show no intercellular substance, and are arranged irregularly in each aveolus. The aveoli open into one another, and are in direct communication with the lymphatics at the margin of the tumour. The fibrous stroma supports well-developed blood-vessels which are quite separate from the epithelial cells.

SCIRRHOUS CANCER.

The scirrhous or hard cancer occurs as a hard round tumour, usually not very large in size, but adherent to the surrounding tissues, and as a rule depressed in the centre, due to contraction of the fibrous tissue. When it arises in the breast this causes retraction of the nipple, an important diagnostic point clinically. On section they present a glistening appearance, from the great amount of fibrous stroma. Yellow patches may be seen here and there, due to fatty degeneration.

Microscopically.—Owing to the slow growth of the tumour, the aveolar structure is exceedingly well marked, and the fibrous stroma is seen to be well developed. The cells in the alveoli are typically those of cancer, but at a late stage in the history of the growth the cells are compressed by the contraction of the stroma. As a result of this they degenerate.

Scirrhous cancers occur in the breast, pylorus, and rectum; less frequently in the œsophagus, testes, ovary, kidney, etc.

ENCEPHALOID CANCER.

This is a tumour of rapid growth, and so the fibrous stroma is poorly developed and the alveoli are very large, but contain a great number of cells; otherwise the microscopic appearance of this growth is the same as described in the scirrhous cancer. They are very soft in consistence, pale pink in colour, and when scraped a great deal of cancer juice exudes. The vegetative power of this growth is very great on account of the number and variety of the epithelial cells. Owing to the vessels having little support, hæmorrhages frequently occur.

The encephaloid cancer often arises as a secondary growth to the scirrhus. As primary tumours they are found growing from the mucous membranes, testes, and breast.

COLLOID CANCER.

There is difference of opinion as to whether colloid cancer ever occurs as a distinct form from the beginning, or is merely a degenerative stage of the other cancers. It is almost confined to the abdominal organs, affecting chiefly the intestines and peritoneum. These cancers are translucent and gelatinous, resembling myxomata in outward characters. The colloid degeneration chiefly affects the epithelial cells. These cells swell, become globular, and colloid material appears in their

interior. In some cells where the process is more marked, a mere film of protoplasm surrounds the colloid globule, and this may ultimately disappear, the colloid material joining the main mass. Thus many alveoli may be seen devoid of cells, their position being occupied by a transparent glassy mass of colloidin. The fibrous stroma, which is frequently affected by the degeneration, is usually scanty.

MELANOTIC CANCER.

This cancer is seldom met with; it is even rarer than melanotic sarcoma. The pigment is chiefly in the stroma, to a less degree in the cancer cells. The tumours may be grey, brown, or black in appearance.

CARCINOMA MYXOMATODES.

This is a form of cancer which assumes a gelatinous texture in consequence of mucoid degeneration affecting the stroma. The cancer cells may likewise partake in the change. The seats of this variety of cancer are much the same as those of the colloid.

TELANGIECTATIC CANCER

Is a rare form of cancer, in which the blood vessels of the stroma undergo dilatation.

CYSTS AND CYSTIC TUMOURS.

A cyst is an excavation bounded by an envelope, and containing fluid or semi-fluid contents. The envelope or capsule usually consists of fibrous tissue, and may be lined with epithelium or endothelium according to its mode of origin.

Simple cysts consists of a single cavity surrounded by a definite wall, and are rounded or ovoid, sometimes irregular in shape.

Compound or complex cysts are those in which a number of diverticula spring from one main cavity. These diverticula may become gradually separated and form small independent cysts.

Multiple cysts consist of two or more simple or complex cysts united together by fibrous or other tissue.

Spurious or **false cysts** may arise as a result of changes in various tumours and other tissues. Thus, in some sarcomas, called "cystic sarcomas," there are numerous spaces filled with serous or blood-like fluid. In such cases the cysts are usually formed either as a result of mucoid softening in the tumour, or of hæmorrhages into its substance.

TRUE CYSTS

Are variously classified by different authors, but a very convenient classification is that according to their origin.

I. **Cysts arising from dilatation of actual or potential cavities or tubes.**—The following may be given as good examples of these forms of cysts—*(a)* On the impaction of a gall stone in the cystic duct, the gall bladder may increase enormously in size from retention of the mucous secretion of its own walls. *(b)* In the condition of hydronephrosis, following obstruction of the ureter, the kidney is converted into a group of cysts. *(c)* Distinct cysts arise from obstruction of the ducts of the sublingual gland, forming the so-called *ranula* in the frenum under the tongue. *(d)* *Wens*, which appear on the scalp, are cysts due to the retention of sebaceous material by an obstructed duct. All these cysts usually have a distinct wall of fibrous tissue lined by endothelium, but their contents usually differ from the secretion of the gland in which they occur. *(e)* When cysts are formed by retention of products in closed cavities they are called "exudation cysts." Bursitis and cystic goître are common examples.

II. **Cysts of new formation.**—The best example of such cysts is the compound ovarian cystic tumour, which is really an adenoma as before pointed out. Other varieties occur in the ovary in connection with the Graafian follicles, and in the broad ligament.

III. **Developmental cysts.**—At an early period of fœtal life, certain organs of the body are composed of tubes, which afterwards become converted into closed cavities. Should the embryonic tubular stage of an organ continue into later life, cysts may result. Thus *(a)* a *congenital hydrocele* arises from imperfect closure of the processus vaginalis. *(b)* *Spina bifida* is due to imperfect closure of the "sinus rhomboidalis," which exists at the lower end of the neural canal in the fœtus. *(c)* *Meningocele* results from imperfect closure of the sutures of the skull, and protrusion of the cerebral membranes. *(d)* The accidental mixture of fœtal structures often gives rise to cysts. The "cystic kidney" is due to the confusion, during development, of the mesonephros (Wolffian body) with the metanephros. *(e)* The "dermoid cyst" is a congenital tumour found about the ovary (commonest position), sacrum, and the side of the head and neck. It may form a tumour of considerable size, partly solid and partly made up of cysts. They contain hair and sebaceous material; but frequently teeth, cartilage, bones, muscle, skin, and all the tissues of which the normal body is made up may be found in them. They are probably due in some cases to the accidental infolding of part of the epiblast in the mesoblast. In other cases they appear to be the result of a blighted ovum becoming ensnared within a healthy one.

IV. **Parasitic cysts.**– A familiar example of this is the hydatid cyst, which will be considered in the study of animal parasites.

PARASITES AND PARASITIC DISEASES.

I. VEGETABLE PARASITES.

All parasitic plants belong to the group "achlorophyllous thallophytes," generally called fungi, which are classified into three sub-groups, viz., *hyphomycetæ* (mould fungi), *blastomycetæ* (yeast fungi), and *schizomycetæ* (bacteria). In disease, bacteria play by far the most important part. The mould and yeast fungi merely exert a local influence.

SCHIZOMYCETES (BACTERIA).

Bacteria swarm everywhere—in the atmosphere, in water, and all over the surface of the earth. They are far more abundant where processes of decay or putrefaction are taking place. The higher in the atmosphere we go, the fewer they become; thus, on a high mountain top we may almost escape from them. They do not normally exist in the blood and tissues of the healthy body, but they are constantly present in the air passages and alimentary canal. In size bacteria are extremely minute, the average being about $\frac{1}{70000}$ in. They are unicellular organisms, consisting of a mass of non-nucleated protoplasm, enclosed in a cellulose envelope. They may be rounded or elongated, rod or thread shaped. Many of them have the power of spontaneous locomotion, and for this purpose they are provided with one or more cilia.

Mode of growth.—Bacteria usually multiply by fission, more rarely by spore-formation. The fission is generally transverse, but may be longitudinal. The spores are formed by a condensation of the protoplasm of the cell, and they lie dormant for some time before they become developed into the fully formed organism.

Classification.—There are two systems of classification adopted—that of Cohn and that of Zopf. Zopf in his system lays stress on the mutability of these organisms, while Cohn, on the other hand, bases his classification entirely on their morphology. Cohn's classification is the more popular. He divides bacteria into **Sphærobacteria** (globular cells), *e.g.*, *micrococcus* or *coccus*; **Microbacteria** (minute rod-like cells), *e.g.*, *bacterium termo*; **Desmobacteria** (larger rod-like or filiform cells), including *bacillum* and *vibrio*; **Spirobacteria** (twisted or spiral cells), including *spirillum* and *spirochæte*. But the above classification is somewhat inadequate, as the observations of Lister have proved, for the same organism, when grown in a different medium, frequently assumes different forms. Thus, when the rod-shaped bacterium (*Bacterium lactis*), which produces the lactic acid fermentation, is sown in urine it assumes thread-like and toruloid forms; when transferred from the urine to "Pasteur's fluid," it takes a rod-like form; and on being re-transferred from the Pasteur's fluid to urine, it assumes thread-like and rod-like forms. Yet throughout all these changes the bacterium still retains its physiological property of inducing lactic acid fermentation. Again, the bacterium which produces butyric fermentation *(Bacillum subtile)*, and the bacterium which produces splenic fever *(Bacillum anthracis)* are identical in morphological characters and life history; yet, when *B. subtile* is injected under the skin of an animal, it gives rise to no evil results, but when *B. anthracis* is injected "splenic fever" ensues. Thus, it seems evident that a classification of bacteria on physiological instead of morphological grounds will ultimately have to be adopted.

The great function of bacteria in nature is to act as so many scavengers, if we might apply the metaphor. By their action the dead bodies of animals and plants are resolved into proximate principles—water, ammonia, carbonic acid, etc.—which can be taken up by plants for their nutrition.

Bacteria are often broadly divided into those which produce putrefaction—*saprogenic* or *septic*, and those which merely set up fermentation—*zymogenic*. The latter live in organic liquids or solids, as milk, cheese, butter, and produce certain chemical changes, consisting in splitting up complex into simpler combinations.

Life conditions of bacteria.—Although some forms of bacteria seem to be able to live on simple compounds of carbon, hydrogen, and oxygen, as, for instance, acetic or lactic acid, yet for their free growth and development they evidently require nitrogenous food. Proteids in an assimilable form constitute the best food for bacteria, and peptonised proteids are especially suitable. "Koch's nutrient jelly" is a very good medium in which to cultivate bacteria; it is a special preparation of macerated meat, to which "agar" jelly is added so that it may remain solid. Aqueous humour, hay infusion, urine, and milk, are all suitable; but there is a great advantage in using a gelatinous medium for cultivating these micro-organisms, as it allows the bacteria to grow in definite forms and produce definite effects upon the jelly, whereby we can readily distinguish the species.

For the cultivation of bacteria a certain range of temperature is necessary. Most of those which produce disease will only grow at a temperature equal to or approaching that of the human body. Bacteria cannot grow under freezing-point, though they may retain their vitality, and all species appear to be killed by a temperature of about 60° C. The spores of some species are, however, not destroyed by heating to boiling-point, but require a temperature of 120° or 130° C. to kill them.

The presence of oxygen is necessary for the growth of some forms of bacteria (these Pasteur called *aërobic*), while others can only grow when oxygen is excluded (*i.e.*, under the surface of the nutritive medium),

anaërobic. But there are some organisms which are capable in certain circumstances of growing under both of these conditions.

Cultivation of bacteria.—As bacteria and their spores exist in the air, great care has to be taken to prevent all forms from entering into the vessel which is to be used, except that special form which is desired for cultivation. Cotton wool allows the entrance of air into a flask, but excludes bacteria, hence plugs of it are used for this purpose. Bacteria fall vertically with the dust, therefore if there be no draught in the room they may be prevented from entering a vessel, provided it has a cover, even though it be otherwise open. If we expose the materials in use to a temperature of 140° C. all germs are killed. A much lower temperature will suffice if the articles are rapidly cooled again, and then reheated, and this process repeated a number of times. A moist temperature of 100° C. is fatal to all germs, therefore the steam of boiling water sterilizes everything. Certain chemicals are used for washing out jars in order to destroy bacteria, the commonest being corrosive sublimate. The rapidity with which bacteria grow is almost incredible ; sixteen or twenty millions may be produced in twenty-four hours from a single specimen. They grow more rapidly in a fluid medium than ina solid one.

The method of isolation commonly employed is that known as "plate cultivation," for which we are indebted to Koch. A tube containing some sterilized jelly is taken, and one drop of tap water (say) allowed to fall into it. The jelly is liquefied by immersing the tube under warm water, while at the same time the drop of water is mixed with the jelly. Then the fluid is poured out upon a glass plate, which is previously sterilized. The fluid flows out into a thin layer, and is allowed to cool and solidify. The plates are then placed under a bell-glass in a moist chamber, and the organisms allowed to grow. After a time, the organisms spring up at different parts of the plate in isolated cultivations, which may be distinguished by their appearance, or recognized with more certainty under the microscope. By means of a sterilized needle, a point of the growth is touched and the organism transplanted into a separate vessel containing jelly. This process may require to be repeated again again in order to finally separate one species, as it is probable that the needle carried away different forms.

Distinctive characters of bacteria under cultivation.—When bacteria are cultivated on a gelatinized medium, they assume peculiar modes of growth,—such as spreading in a branching form, or merely showing points. Various colours are produced according to the species. Some cause the medium to liquefy. Others cause bubbles of gas to be evolved. But the chief method of detecting species is by the different reactions they yield with aniline dyes, as seen under the microscope.

Agents which affect the growth of bacteria.—When bacteria are arrested or hindered in their growth by the contact of any substance, what is known as "antisepsis" or antiseptic action takes place; and the agent employed is called an antiseptic. Should, however, the organisms be not only arrested in their development, but actually killed, then "disinfection" ensues; and an agent which can accomplish this is termed a disinfectant. Many antiseptics have no disinfecting value, while some admirable disinfectants have practically no antiseptic value, *i.e.*, if diluted until they are not powerful enough to kill bacteria, they will neither hinder their development. After elaborate experiments on various forms of bacteria with different chemicals, Koch arrived at the following conclusions,—a solution of corrosive sublimate of 1 in 20,000 killed the spores in ten minutes; 1 in 300,000 arrested their growth. A 5 % solution of carbolic acid killed the spores in twenty-four hours, while 1 in 400 arrested their growth. Although corrosive sublimate stands highest as both a disinfectant and antiseptic, yet it is worth noting that carbolic acid has a special power of preventing the development of certain organisms which are especially related to suppuration. Boric acid, borax, salicylic acid, camphor, and eucalyptol were found to possess little antiseptic or disinfectant action. Even sulphurous acid in a 1 % solution had very little effect on these organisms, and could only act on the surface of the media, having no power of penetration.

Relations of bacteria to the living body.—During perfect health, the blood and tissue are free from bacteria. But they are found to be always present throughout the alimentary canal, in the respiratory passages, on the skin, and even in the glands of the skin. What becomes of all these organisms? The greater number undoubtedly pass out of the body again. It is hardly possible that any can penetrate into the deeper tissues through the uninjured skin. Those which settle on the mucous membranes are for the most part not absorbed, but are thrown off after a longer or shorter time. But, as experience has shown that a certain number of bacteria can be absorbed from the mucous membranes, one must ask the question—how are the tissues of the body kept free? It appears from recent observations that the activity of the cells, and especially the leucocytes, prevents the growth of bacteria in the body. Metschnikoff, in his experiments on frogs, found that the leucocytes absorbed and apparently destroyed anthrax-bacilli artificially introduced into their bodies. Leucocytes, after absorbing bacteria, hypertrophy; so it is possible they absorb and assimilate these organisms.

Evidence required to prove any disease to be caused by bacteria.—Koch lays down the following conditions as necessary to the establishment of proof that a special micro-organism is the cause of any particular disease. (1) "The micro-organisms must be constantly found in the blood or tissues, or both, of the men (or animals) suffering

from the disease. (2) The micro-organisms must be got to grow in some medium outside the body, and by successive cultivations, completely separated from all matter belonging to the body from which they came. (3) After being thus cultivated through several generations, the organism must be inoculated into some animal, and reproduce in it the original disease. (4) In the tissues or blood of this animal the same micro-organism must be found which was found in the original patient." But the great difficulty in fulfilling these conditions is, that there are some human diseases which are not known to affect the lower animals and which cannot be communicated to them, so that inoculation experiments do not always succeed.

The **immunity of species** to the action of bacteria varies much. Thus there is an organism *micrococcus tetragenus* (*i.e.*, a micrococcus forming tetrads or groups of four), which is found in the walls of cavities in the lung in cases of *phthisis* in the human subject. When cultivated and inoculated into white mice, it produces death in from three to four days. On the other hand, common brown mice have immunity. On rabbits and dogs the same organism has no effect ; but in guinea pigs it produces the same effects as in white mice, though sometimes it leads to the formation of local abscesses.

Again, "*anthrax*" is a common disease among cattle and sheep, the specific organism which gives rise to it being *Bacillus anthracis ;* but Chauveau inoculated certain sheep from Algeria with this bacillus and no untoward phenomena ensued.

Immunity of species may be innate or acquired. Innate or congenital immunity is well illustrated in the case of the negro race, which never suffers from yellow fever. This congenital immunity may be explained by the disease having acted on the race for many generations, during which time it will have weeded out the most susceptible individuals. "Acquired immunity from any disease is generally the result of having had the same disease before, either in its ordinary or modified form. In the majority of specific diseases, one attack protects against another for some years, or even for the whole of life ; and there is no such complete immunity as in the condition which immediately follows an attack." Thus, if a person suffers from small-pox, he is not liable to a second attack. The same results may sometimes be obtained by subjecting the body to the action of the poison in a modified form, *i.e.*, by vaccination. Greenfield found that after animals were inoculated with the bacillus of anthrax they became insusceptible even to large quantities of the virus. The organism of "*mouse septicæmia*," if inoculated into the ear of a rabbit, produces a form of erysipelas. After repeated inoculations the same condition cannot be set up, the animal having acquired the power to resist the energy of the bacillus. But, on the other hand, diptheria rather favours the occurrence of a second attack ;

and frequently families are met with who are liable to a recurrence of scarlet fever and measles.

Entrance of bacteria into the body, and their mode of action.—Inoculation of bacteria usually takes place at a free surface, such as a wound, abrasion or excoriation of the skin; this is especially the case if there be any necrosed tissue present, which may form a nidus for their growth. Such is the mode of inoculation in malignant pustule, syphilis, tubercle, pyæmia, and puerperal fever. It is probable also that a similar mode of entrance is effected by the organisms of some alimentary diseases — typhoid and tubercular ulceration in the intestine. Bacteria may find entrance into a living body, but owing to the high vitality of the tissues, be unable to produce any damage.

"Bistournage" is the name given to an operation on animals, sometimes adopted instead of castration. It consists in twisting the spermatic cord so as to sever the blood-vessels from the testicle, and in that way to produce atrophy of the organ. Chauveau performed a series of experiments on bistournage, and found that under ordinary circumstances the testicle simply atrophied. But, if he injected into the blood a quantity of putrescent material, instead of having a slow process of necrosis in the testicle, there occurred an intense suppurative inflammation.

If some of the organisms which have the power of producing suppuration be introduced into the system of a healthy animal, they produce no effect; but, if the wall of a vessel be artificially damaged, they will attach themselves to the vessel wall, propagate there, and enter the blood.

Many bacteria have the power of remaining dormant in the body for a very long time. This has been shown from observations on tubercle, leprosy and glanders. Any damaged part, such as a caseous patch, favours this condition; the organism remaining in such a patch for years, when, as the result of a stimulus, it suddenly begins to show signs of growth.

Ptomaines or cadaveric alkaloids are products resulting from the decomposition of proteids; but that these products are due to the action of bacteria has not yet been proved. We sometimes meet with cases of poisoning by tinned meats and other kinds of food which have undergone this form of decomposition. "Ptomaines are powerful functional poisons, especially to the nervous system. They have not the destructive action on tissues characteristic of ferments, nor have they necessarily any irritant action" (Payne).

DISEASES PROVED OR BELIEVED TO BE CAUSED BY BACTERIA.

SIMPLE ACUTE SUPPURATION.

This condition was discussed under inflammation, to which the student is referred.

SEPTIC DISEASES.

Septic diseases are divided into certain forms under the name of **Septicæmia,** in which there is no definite suppuration present; and a number of forms called **Pyæmia,** in which there is present some localized suppuration giving rise to secondary foci of suppuration in distant parts.

(A.) SEPTICÆMIA.

This frequently occurs after parturition, when it receives the name of "puerperal fever." The condition sometimes follows the poisoning of wounds. The entrance of the poison may be through an almost imperceptible puncture, such as caused by a dissecting wound.

Phenomena which are more or less common to the various forms. They present variety both in acuteness and in the character of the symptoms. All the symptoms of severe fever are present,—high temperature, frequent pulse, great prostration, and usually delirium. Sometimes, however, the temperature is subnormal, when the condition is called "septic toxæmia." Hæmorrhages frequently take place from the mucous surfaces. If the patient recovers there are no after effects produced. Death, however, frequently results from the disease, and it may even take place a few hours after inoculation; on the other hand it may be more prolonged.

Post mortem the body usually undergoes rapid decomposition. The blood in the vessels is remarkably fluid, dark in colour, and coagulates imperfectly. But if the decomposition sets in slowly, the blood may be normal. Minute hæmorrhages are to be observed on the serous membranes, especially those of the pleura and pericardium. The spleen is swollen, soft, and diffluent, more rarely it is pale and greasy. In the liver and kidneys the epithelium has undergone cloudy swelling, and the kidneys may even be broken down and quite granular. Cloudy swelling is also constant in the heart muscle. Microscopically, there are usually bacteria found in the blood, and frequently also in the tissues. But there are cases recorded where bacteria were not observed after the most careful examination of the blood and tissues.

(B.) PYÆMIA.

Pyæmia cannot be definitely separated from septicæmia, but the name is applied to a more chronic state of blood-poisoning, associated with the production of secondary abscesses. The condition usually follows a putrid wound, but it may also follow an internal suppuration, such as that seen in the middle ear. There may not be anything in the external character of a wound to arouse suspicion that septic matter is being absorbed, but there is usually a deficiency of healthy granulations and free suppuration, which apparently act as a barrier to the introduction of foreign bodies.

Phenomena.—Pyæmia frequently commences insidiously; at other times it is ushered in by a sudden and severe rigor, the temperature rising five or six degrees in a few minutes, and falling again in the space of half-an-hour, with profuse perspiration and a sense of the most complete exhaustion. Rigors are seldom absent. "The general symptoms are those common to all forms of blood-poisoning, only they are seldom so acute as in septicæmia" (Moullin).

Post mortem the appearances are almost the same as those of septicæmia, with this addition, that we find secondary abscesses in various organs. These abscesses are most common in the lungs, and vary in size from a pea to a hazel-nut. In the substance of the lung they are rounded; towards the surface they are conical in shape. They are always surrounded by a zone of intense injection, and contain a greenish offensive pus, with debris of lung tissue. In appearance they look like pulmonary infarcts which have undergone central suppuration. If the abscess is near the surface of the lung there may be a suppurative pleurisy set up. The abscesses are commonest at the lower or posterior parts of the lungs. The cortex of the kidney, liver, heart-wall, and brain substance are all common sites for such abscesses.

Method of production of secondary abscesses.—The wound from which the disease starts is putrid, and so forms a nidus for the growth and development of bacteria. The bacteria penetrate the surrounding tissues and invade the walls of the nearest vein. As a result thrombosis takes place in the veins; but the bacteria enter the clot and decompose it, so that it breaks down into a substance resembling pus. This soft mass of altered blood-clot, which is green in the centre, may plug the lumen of the vein for a considerable distance. As a result of these changes the wall of the vein becomes intensely inflamed (*i.e.*, it is in a condition of *phlebitis*), and gangrene may even set in. Portions become detached from the decomposing clot, and are carried into the blood stream. They travel to the right heart, and thence proceed until they become impacted in one or more of the smaller branches of the pulmonary artery (constituting embolism). The embolus in this case is septic, and hence the infarcted area undergoes rapid softening and decomposition, resulting in an abscess (embolic abscess). In the liver, abscesses are found when the source of pyæmia is in the intestinal tract, the septic matter being conveyed by the portal vein. Those arising in the kidney and brain may be explained as follows:—When a secondary abscess starts in the lung the vein corresponding to the infarcted area is filled with a decomposed clot, from which portions may be detached, reach the left

heart, and enter the general circulation. But with this explanation there would necessarily be always a secondary abscess in the lungs whenever there was one in the brain or kidney, so that the above does not always hold, as we may find an abscess in either of the latter positions without the lungs being affected. "Experiment has proved the possibility of finely divided material passing through the pulmonary capillaries and becoming arrested in the systemic." The secondary lesions will therefore be in capillaries, and not in larger vessels

It is not uncommon to have suppuration in joints in cases of pyæmia. The suppuration usually begins in the tissues round a joint, and secondarily opens into the synovial cavity. We may also have suppurative inflammation of the various serous membranes, especially those of the brain and pericardium.

Medical pyæmia is a term applied to the disease when it arises without any discoverable external wound. These cases may be regarded as showing the reception of the poison by some other channel than by a wound, although a few authorities are disposed to think that the pyæmic poison may be spontaneously generated within the body.

The organisms which are found in pus during ordinary inflammation are *Staphylococcus pyogenes aureus*, *Staphylococcus pyogenes albus*, *Staphylococcus osteomyelitis*, and *Streptococcus pyogenes*. If the pus or tissues be examined in pyæmia the above forms of bacteria are found, but by far the most common form is that of *Streptococcus pyogenes* and, next, *Staphylococcus pyogenes aureus*. The question then arises, why are the effects of pyæmia so grave compared with those of ordinary suppuration? It would appear to be due either to the production of some albuminous substance, or ferment produced by the bacteria, this ptomaïne or toxine giving rise to the fever and poisoning; or else simply to the number of bacteria present, since, under ordinary circumstances, the micrococci of suppuration enter the blood, if at all, in very small numbers.

ANTHRAX.

SYNONYMS—SPLENIC FEVER: SPLENIC APOPLEXY.

This is primarily a disease of cattle, but not infrequently is communicated to man. In cattle it occurs as an acute infectious disease, often proving fatal. It affects also sheep, horses, and deer, and can be communicated by inoculation to many other animals. The poison is known to be a special form of bacillus—*Bacillus Anthracis*. It is rod-shaped, and very much larger than the average bacterium, so that it can be easily studied. It retains its vitality for a long time, even many months after being dried up. In the case of man, anthrax is almost invariably communicated by direct contagion; hence it is most frequently observed in those who deal with animals—veterinary surgeons, farmers, &c., or in persons engaged in working among raw wool or horse hair. It may occur either in an external local (malignant pustule), or internal form.

Malignant Pustule occurs chiefly on exposed parts—the face and arms. It commences as a small raised pimple, usually slightly irritable. This pimple rapidly spreads until it reaches a quarter of an inch in size, when a vesicle appears in its centre. The inflammation proceeds outwards, and at the same time more vesicles arise at the periphery of the swollen patch. At a later stage the central vesicle dries up, and leaves a brown edge ; when the swelling is about an inch in diameter, sharply raised, with a ring of vesicles round it, and a brown scar in the centre. In the region of this pustule there is usually intense œdema, and the lymphatic glands are enlarged. The condition is usually unaccompanied by general symptoms, but poisoning of the system may occur and lead to a fatal result. The bacilli are chiefly contained in the vesicles, but they penetrate into the deeper parts along the lymphatic spaces. The intense inflammation leads to an enormous inflammatory engorgement of the subjacent tissues, and the superficial parts undergo necrosis, producing a black central eschar.

Internal anthrax (wool sorters' disease) may affect the respiratory or alimentary canal. In the case of the alimentary canal it affects either the stomach or intestines, or both. It produces a diffuse inflammation of the submucous tissue, which frequently ends in death. In the respiratory passages it occurs in the bronchi and lower part of the trachea. There is great swelling of the mucous membrane, and hæmorrhages take place into the mucous and submucous tissues. The process is accompanied by great swelling of the mediastinal and bronchial glands. In these glands, and in parts of the surrounding tissues, numbers of bacilli are to be found. In cows, the spleen is enormously enlarged, but this is not the case in man.

OTHER DISEASES ASSOCIATED WITH SPECIFIC BACTERIA.

Tetanus (lockjaw), when occurring in connection with wounds is associated with some specific virus. The same holds with some of the acute fevers, such as **scarlet fever, measles**, and **smallpox** ; but though it is highly probable that the organisms which set up these diseases are bacterial in nature, yet they have not been isolated.

But the condition is different in the diseases of **cholera, typhoid** (enteric fever), **relapsing fever, diphtheria**, etc., where specific bacteria have been isolated in each case. Though it has not been absolutely proved from experiments that each of these conditions is directly caused by the bacterium found, yet the hypothesis is highly probable.

CHRONIC INFECTIVE INFLAMMATIONS DUE TO BACTERIA.
(INFECTIVE GRANULOMATA).

The term "infective granulomata" includes the diseases *tubercle*, *syphilis*, *glanders*, and *leprosy*; and the group possesses the following general characteristics. (1) They are infective, *i.e.*, may be inoculated from one animal to another. (2) They set up inflammatory processes which result in the formation of granulation tissue. This granulation tissue has certain special chararacters, which differentiate it from ordinary granulation tissue as seen in wound healing. *(a)* It does not rise from a definite local irritation; *(b)* as a rule it does not tend to the formation of distinct cicatricial tissue; *(c)* it has a great tendency to persist, and is not absorbed on the removal of the irritation; *(d)* it has a marked tendency in all cases to undergo degenerative processes, which often occur very early. (3) They tend to spread throughout the body when inoculated.

TUBERCULOSIS.

Tuberculosis accounts for over 10 per cent of the mortality of Great Britain. The condition is not limited to man, but affects also bovine animals, rodents, poultry, etc. It is a universal disease among men, though there is strong evidence that it has spread to some races within comparatively recent years. Tuberculosis includes a variety of diseases, which receive particular names according to the organ affected. Thus, in the lungs it produces *phthisis* or consumption; in the skin—*lupus*; in the bones—*caries*; in the joints—*white swelling*. The condition may be acute or chronic, local or general. It generally begins as "localized tubercles" in the lung or intestinal tract, but being infective, when once started it may spread throughout the body by the lymphatics or blood-vessels.

That tuberculosis is produced by a definite form of bacillus known by the name of *Bacillus tuberculosis* there is no doubt, as all the conditions necessary for such proof (see page 60) can be in this instance established. The B. tuberculosis is one of the smallest bacilli; it consists of a motionless rod about $\frac{1}{3}$ or $\frac{1}{4}$ the diameter of a red blood corpuscle in length. It is distinguished from all others (except the bacillus of leprosy) by certain relations to aniline dyes. It absorbs these colours more slowly than some other organisms, but retains them with greater tenacity, and when afterwards treated with a dilute mineral acid it remains unchanged; whereas any other bacteria so stained are deprived of their colouring, hence this is a differential stain. The organism sometimes shows a beaded like structure, which has led to the supposition that its mode of growth is by spore-formation. The cultivation of tubercle-bacillus is rather difficult as it will not grow in nutrient jelly. Sterilized solidified blood serum is perhaps the best medium for its development; and if kept at a moderately warm temperature the bacilli begin to make their appearance on the surface of the serum in from ten to fifteen days. The bacillus is found in tissues where the disease is most active, but it is also found in the blood, and in the excreta of organs which are not themselves affected by the disease. Thus, in phthisis it has been shown that the organism may be present in the fœcal excreta, and the milk from a cow may contain it although the udder is not affected. When introduced into an animal, the bacillus multiplies and spreads through the surrounding tissues, chiefly along the ordinary lymph spaces and channels. The organisms may remain dormant in certain situations, even for years, without producing any other than a direct local effect.

There is no doubt that the bacillus acts like a foreign body in the tissues, and the specific irritation may lead to one of two results,—production of tubercular granulations, or destructive inflammation attended by caseation.

I. **Tubercular granulations** (tubercle follicles) are seen with the naked eye as small nodules of a grey gelatinous appearance. They vary in size from almost invisible points to about an eighth-of-an-inch in diameter. They are either scattered irregularly or grouped into small masses. As they become older, these nodules turn yellow in the centre and tend to calcify, while the margins become fibrous.

Microscopically, each nodule is seen to be made up of one or more centres of growth, which are termed "tubercle follicles." Each tubercle follicle has a definite and regular structure,—in the centre is a large irregular multi-nucleated mass of protoplasm, the so-called "giant cell"; outside this there is a series of large round cells called "epithelioid," from their resemblance to epithelial cells; outside this again, we have small round connective tissue cells; and if the disease be very chronic a kind of capsule of fibrous tissue encloses all. There are no new blood-vessels in this granulation.

Formation of the giant cells.—Microscopic examination of the lung has shown that giant cells are commonly derived from a single epithelial cell, which undergoes great enlargement. The nucleus divides and gives origin to the many nuclei of the giant cell. It is probable that giant cells may also be formed by the coalescence of several epithelioid cells.

The following changes may take place in these granulations—(*a*) fatty degeneration and caseation; (*b*) calcification; (*c*) they may soften and form pus (tubercular abscess).

Should the disease assume an acute form, there are no tubercle follicles to be seen, but a—

II. **Destructive inflammation attended by caseation.** In this condition there are no naked eye characteristics. It is very well seen in the lymphatic glands, and in the lung in acute phthisis. In the lung

the alveolar walls are thickened, the small air channels are filled with cellular elements. In this mass of cells, staining reveals numerous tubercle bacilli.

MODE OF INFECTION AND SPREAD OF TUBERCLE.

Infection may occur from the sputum of phthisical patients. On the sputum drying the tubercle bacillus may be blown about, and since it retains its vitality when dried, and has the power of resisting various disinfecting agents, it is easy to understand how tuberculosis may be conveyed from one subject to another. The commonest mode of infection is by the air passages, and the apices of the lungs are specially liable to early infection. The reason why the apex is so often first affected probably depends on the fact that respiration is less active there than in any other part of the lung, and therefore the bacillus cannot be so easily expectorated. In children, we frequently find caseous tubercular glands in the neck, which may be set up by the bacillus lodging in the crypts of the pharynx and spreading by the lymphatics. Children are very liable to be affected in the abdominal organs, the disease manifesting itself first in the mesenteric glands. This may be accounted for by the introduction of the bacillus with food. Milk is the food which is most apt to be contaminated with the tubercle bacillus, the disease being so very common among cows.

Incubation.—The organism may remain dormant for a long time and merely set up a local inflammation in a few glands, as exemplified sometimes in the enlargement of glands in the neck, after which the whole body may become affected from some additional cause. This additional factor may be depressed vitality on the part of the patient, or increased nutrition to the bacillus; no doubt both are frequently combined.

Heredity.—If tuberculosis be hereditary, as is commonly maintained, it can hardly be said to be the result of bacteria. One would perhaps be more correct to say that there is an inherited predisposition to the disease, as exemplified in an individual with a congenital weak flat chest, or having a tendency to bronchitis, thus rendering such a patient more liable to attacks of tubercle. But there is a possibility of direct infection from the mother through the placenta. There is sufficient evidence to prove that cats can affect their offspring through the placenta. It has even been said that direct impregnation of the ovum may take place, but this is doubtful. The tubercle bacillus has, however, been found in the testicle and vesiculæ seminales when tuberculosis was entirely absent from all other parts. But the disease may obviously be transmitted from the mother to the child in various ways, as, for example, through the medium of the milk.

Diseases which predispose to tuberculosis.—Diabetes and anæmia favour the occurrence of the disease. Persons affected with acquired syphilis are very liable to tuberculosis. On the other hand, a venous condition of the blood is very unfavourable to the development and spread of tubercle bacillus.

LUPUS

Is simply a localized tuberculosis. It affects the skin of the face chiefly about the nose and angle of the mouth. The **morbid anatomy** consists in an infiltration of the corneum, which extends into the subjacent tissue. The granulation tissue is much more vascular than in other forms of tuberculosis, and it tends to early desquamation. In chronic cases giant cells are formed, which have small masses of granulation tissue in relation to them, closely resembling tubercle follicles. The tubercle bacillus has been found in this tissue, and after cultivation and isolation has produced tuberculosis on inoculation (Koch and Cornil).

The condition leads to a nodular thickening of the skin. This spreads with a variable amount of inflammation, and there ensues either a simple atrophy of the thickened tissue, or ulceration. The destruction of the skin and subcutaneous tissue may be very extensive, patients commonly losing a large part of their nose or mouth.

Some forms of lupus are, however, not tubercular; they may be scrophulous or syphilitic. Of course such cases have not the typical granulation tissue of tuberculosis.

LEPROSY (ELEPHANTIASIS GRÆCORUM).

Leprosy, which was so very common throughout all Europe in the middle ages, is now almost restricted to Sweeden, Norway, and some parts of Russia. It still exists in the east—India, Syria, and Egypt. According to its pathology the disease is divided into two forms, viz.,—(1) tubercular or cutaneous, (2) nervous or anæsthetic.

I. The **Tubercular** form is characterised by the formation of nodes or diffuse infiltrations into the tissues. These, when cutaneous, are usually seated on the surfaces exposed to the air—the face, hands, and feet. The skin becomes greatly thickened and thrown into irregular folds, so that the features of a subject may be entirely obliterated. The nodes may reach the size of a walnut, and their basis is made up of cellular granulation tissue lying beneath the epidermis. This tissue contains a number of large cells, which may be as large as the giant cells of tubercle, but do not contain so many nuclei, usually not more than two ("lepra cells"). When the nodes break down, leprous ulcers are formed. The nodes of the mucous membrane are more apt to ulcerate than those of the skin; common situations being the inside of the nose, and in the conjunctiva, mouth, and larynx.

"Scrofulus" is an obsolete term
It is practically the same as "tuberc

II. **Anæsthetic** form.—In this form the leprous growths occur in the nerve sheaths, leading to distruction of the nerve functions. There is therefore local amyotrophy and anæsthesia.

The two forms above mentioned are always more or less associated with one another in the same patient; but sometimes the one and sometimes the other is more marked. Leprosy may involve almost any structure of the body. It is practically an incurable disease. Hansen and Neisser have discovered that leprosy depends on the presence of a specific bacillus *(Bacillus lepræ)*. The bacillus is found in all leprous foci, generally enclosed in the larger cells. That the disease is hereditary has not yet been proved. It is however contagious, for on being introduced into the Sandwich Islands about thirty years ago, it became extremely virulent there.

GLANDERS AND FARCY.

These are forms of a contagious disease prevalent among horses and which is communicable to man by direct transmission.

Glanders consists in an inflammation of the mucous membrane of the nose, like a purulent catarrh. This is accompanied by the formation of oval or rounded elevated patches, which break down superficially and produce shallow ulcers. But the ulceration may extend deeply and destroy the bone. From these ulcers there is a good deal of ichorous discharge. The disease spreads to the lymphatics in the neck, the glands becoming inflamed and undergoing caseous degeneration. At a later stage ill defined nodular masses appear in the lungs.

In **farcy** the superficial lymphatics and skin are affected, and it is of a more chronic nature than glanders, into which it sometimes passes. It usually affects the hands or the arms. It produces cutaneous or subcutaneous nodules, sets up inflammatory thickening of the lymphatics, and frequently causes the formation of abscesses in the glands or their vessels.

In **man** the disease is nearly always acute. It does not necessarily attack the nasal mucous membrane, for frequently there occurs a pustular eruption on the face, which is followed by a speedy ulceration. The eruption may be all over the body. The lungs are the chief internal organs affected, where a number of acute abscesses are formed. In man this disease has a great resemblance to pyæmia. The bacillus of this disease has been cultivated and inoculated into rabbits, guinea-pigs, and horses, with the result of reproducing the condition.

SYPHILIS.

This disease may be considered a perfect type of an infective disease, produced by a virus introduced from without; and although the special organism has not yet been discovered, syphilis is included among the "infective granulomata," as it has all the characteristics of this group. After inoculation, there are definite local changes of an inflammatory nature. Then there is a subsequent infection of the system, locally, first of all, by the lymphatics, and, later, generally both by the lymphatics and blood-vessels. At a later stage there is a formation of slowly growing granulation which is characteristic of this disease.

The original spot of inoculation shows the "primary chancre," which is an ulcerating node distinguished for its hardness. When examined under the microscope the corium shows changes of an inflammatory nature. There is a large number of round cells, some epithelioid cells, and generally a few giant cells. The structure has not the definite form of the tubercle, and it is much more vascular. The hardness depends on the thickening of the connective tissue fibres, as well as on new formation of tissue. The development does not advance beyond this condition; the greater part of the affected tissue breaks down and ulcerates, or is re-absorbed. Some part of the cells may go to to form a cicatrix.

Syphilis may be either *congenital* or *acquired*, and although the phenomena are somewhat similar in both conditions, yet they are fully better marked in the inherited form. In congenital syphilis the changes are not only more intense, but occur with greater rapidity than those in the acquired variety.

Symptoms of congenital syphilis.—From one to two months after the birth of the child there may be no signs of disease. But soon a slight roseolar eruption makes its appearance; this is soon followed by a more severe eruption, which has the the characters of "rupia." At a later period more characteristic symptoms occur. The infant snuffles in breathing, owing to persistent inflammation of the nasal mucous membrane. The angles of the mouth become inflamed, which leads to the formation of radiating scars, and mucous patches are found inside the mouth. The temporary set of teeth, being advanced in calcification, are not affected in shape, but the permanent teeth become peg-shaped, and exhibit a central notch. This is best seen in the upper median incisors. Between the ages of fifteen and eighteen the head assumes a peculiar shape, and about the same period interstitial keratitis (inflammation of the cornea) makes its appearance. Deafness, resulting from chronic inflammation in the ears, is also a common symptom.

Of the internal organs, the effects of this disease are most marked in the liver. The organ is greatly enlarged, and shows a diffuse fibrous change throughout its substance. The normal liver cells atrophy and undergo degenerative processes. Similar changes take place in almost every other organ, especially the heart and lung.

Syphilitic infiltrations lead to the thickening and contraction of mucous membranes, so that the lumen of mucous passages becomes lessened. Thus, the larynx or intestines may be narrowed, and ulceration frequently sets in.

Syphilitic gummata consist of nodular masses of granulation tissue. They are hard and firm, and at first not well defined. They have an outer fibrous zone, which passes indefinitely into the surrounding tissue. The centre is yellow, and if the gumma has lasted for some time, caseating degeneration may set in. **Microscopically** the mass is seen to be made up of fibro-nucleated tissue. If the gumma is not recently formed, the central portions consist of a granular or homogeneous material. The outer portions show more perfectly formed fibrous tissue, and are vascular. The process appears to begin in a growth of tissue in and around the smaller arteries. The necrosis of the mass is therefore due to total arterial obstruction.

Gummata may occur in any of the internal organs. They are very common in the liver, brain, and bones. In the liver of an adult a gumma is usually found on the surface of the organ, enveloped in a fibrous capsule. The central portion of a gummatous mass may become absorbed, leaving only a hard fibrous scar to mark its position.

Syphilitic endarteritis.—This change consists in a great thickening of the inner coat of a vessel by the formation of granulation tissue, which grows from the endothelium and connective tissue of the part itself. As a result, the lumen of the vessel becomes narrowed, and thrombosis usually sets in. The peculiarity of this change is, that it occurs quite apart from any external irritation, and therefore must depend on the direct action of the syphilitic virus upon the inner coat of the vessel. The outer coat of the vessel is usually affected also, nodular thickening being found around it. Syphilis has a peculiar tendency to produce degenerative changes in large vessels also, and thus aid in the occurrence of atheroma. It is one of the chief factors in the production of aneurism of the aorta.

Syphilis is one of the diseases which leads, without the occurrence of suppuration or chronic discharge, to waxy degeneration. The kidney is the organ most frequently affected with waxy disease in syphilis.

The heredity of syphilis.—Infection more frequently takes place from the father than from the mother. The mother is generally infected also, but it is usually limited to her reproductive organs. Direct contagion from the mother frequently takes place, as the maternal part of the placenta is commonly the seat of syphilitic changes. There is great probability that the fœtus may be inoculated in utero. The time when the eruption makes its appearance in a syphilitic child would lead one to conclude that inoculation had taken place at, or shortly before, birth.

ACTINOMYCES or RAY-FUNGUS.

The botanical position of this parasitic plant is still a matter of uncertainty. When cultivated it grows in round masses which vary in size. **Microscopically** these masses appear to be composed of a number of radiating threads with swollen, club-shaped ends, forming a rosette This variety of fungus produces the disease called *Actinomycosis* in cattle, and to a less extent in man.

ACTINOMYCOSIS.

In **cattle** this disease usually begins in the mouth in the form of tumours of the jaw. It may lead to the formation of abscesses in the jaw, and then spread to the neighbouring lymphatic glands. At a later stage the tongue and mucous membrane of the mouth become affected, hard nodular masses being formed; producing the so-called "woody tongue." It may spread to the pharynx and larynx, and nodular masses have even been found in the lungs. These masses tend to soften and undergo suppuration. "When not softened they show the structure of a typical tubercle with giant cells, epithelioid cells, leucocytes, and fibroid tissue, the actinomyces fungus acting as the centre of irritation. The disease is commonest in horned cattle, but may affect swine and horses. The carnivora appear to be exempt" (Payne).

In **man** the disease is very rare, not more than 40 or 50 cases being known altogether, and of these, few have happened in this country. The neighbourhood of the jaws and neck are the commonest parts affected, where chronic deep seated abscesses are formed. Sometimes it starts with disease of the vertebræ, forming a retro-pharyngeal abscess. Less frequently it begins in the lung or liver. According to Israel, when the disease appears in the jaw, the fungus enters by the mouth; when it occurs in the lung, the fungus enters by the respiratory tract; and from the intestinal tract the organism may find its way to the liver. All the cases so far observed in this country have been abscesses of the liver, and proof of the condition can only be established by the discovery of the actinomyces.

Nodular tumour-like growths characterize the disease in cattle, while in man there is always a tendency to breaking down and suppuration.

HYPHOMYCETÆ or MOULD FUNGI.

The Hyphomycetes are the well known fungi which form the flocculent covering or pellicle seen on decaying organic matter, and receives the familiar names of mildew, mould, etc. The mould fungi are characterized by the formation of a *mycelium.* Each mycelium consists of a number of filaments called hyphæ. These hyphæ are generally hollow, and divided by septa. The organs of fructification consist in

modifications of a number of hyphæ into spherical, ovoid, or cylindrical cells, often strung together into a kind of chaplet. These cells are called *spores* or *gonidia*.

The commonest and most important of these organisms are *Aspergillus fumigatus* and *Mucor mucedo*. If the spores of these fungi be injected into the blood of an animal in large numbers, they produce toxic symptoms.

FAVUS.

Favus, or tinea favosa, is a disease commonly affecting the scalp. The condition is produced by the fungus *Achorion Schœnleinii*. This organism first attacks the hair and hair follicle, after which it spreads on the surface of the skin. It leads to the formation of light yellow, dry, cupped crusts, which have a peculiar mousey odour. The crusts consist of altered epidermic cells, in which there is a mycelium and spores.

PITYRIASIS VERSICOLOR.

This is a skin disease which most frequently attacks the back and chest. It is most frequently met with in those suffering from wasting diseases, especially if they wear flannels next the skin, and seldom wash. If the yellowish-brown flakes be scraped off the skin and examined microscopically, curved mycelia are observed, along with small, round or oval bodies which arrange themselves in groups. These latter are the spores of an organism which has been named *Microsporon furfur*.

RINGWORM.

This condition includes **tinea circinnata** or ringworm of the body; **tinea tonsurans** or ringworm of the scalp; and **tinea sycosis** or ringworm of the beard and other hairy portions of the face. All these forms of this disease are produced by one fungus—*Trichophyton tonsurans*. This parasite consists of long slender jointed filaments (mycelium) which are usually more abundant and more finely branched than in favus. The spores are small, round, and highly refractive, and usually very abundant. These spores infiltrate the hair bulb, while the mycelia spread up the shaft of the hair.

BLASTOMYCETÆ or YEAST FUNGI.

Yeast fungi have a very important physiological function in causing fermentation of sugar—splitting it up into alcohol and carbonic acid. They form the yeasty scum which appears on the surface of alchoholic liquors. Unlike the moulds, the yeasts have no mycelia. They are composed of cells, rounded or ovoid in shape, and grow partly by a process of budding from the old cells, and partly by the formation of spores. Their relation to disease is not very important.

THRUSH.

Thrush, or Aphthæ, affects children who are fed from dirty bottles. It consists in the formation of white patches on the mucous membrane of the mouth. Sometimes the alimentary canal is similarly affected. The fungus present on these white patches is *Saccharomyces Albicans*, formerly called *oidium albicans*.

II. ANIMAL PARASITES.

I. TREMATODA OR FLUKES.

These are soft worms, usually flattened or tongue-shaped. They possess pores or suctorial openings, by means of which they adhere to surfaces. The intestinal canal is not pervious, there being only an oral orifice. They spend their life partly in an "intermediate host," and partly in a "final host."

DISTOMA HEPATICUM.

The "liver fluke" is about one inch long by half-an-inch wide. It is leaf-shaped, the broad end projecting like a beak to form a kind of head. The head-end bears a small suctorial disc, in the centre of which is the oral orifice. At a little distance behind this on the ventral aspect is another suctorial disc, and between these discs is a small opening—the genital orifice. The discs are organs of locomotion and adhesion. The intestinal canal bifurcates. These creatures are hermaphrodite. The uterus lies behind the posterior disc, and consists of a convoluted tube. Towards the hinder part of the body lie the ovaries, and between them are the bifurcated testes. The body of this parasite is covered by a thin chitinous cuticle.

The adult animal infests the biliary ducts, and more rarely the intestine and vena cava. This parasite produces the "rot" in sheep, and although it is common among ruminants, it is rarely found in man.

Cycle of development. From the egg there escapes a small ciliated free-swimming embryo. This embryo fluke penetrates the body of the intermediate host, which is the fresh water snail (Lennæus), and becomes encysted. By internal gemmation, this encysted creature gives rise to a large number of intermediate forms called *rediæ*. These rediæ leave the sporocyst and find their way to the liver of the snail. Within each of them is formed a tailed larva or *cercaria*, which leaves the redia as it becomes mature and passes out of the body of the snail. This tadpole-like creature swims about until it is ultimately taken up by its final host, and passes to its habitat by boring through the wall of the alimentary canal.

DISTOMA LANCEOLATUM,

As its name implies, is lancet shaped, and in structure is very similar to the above species, but much smaller. Its head does not project, and the integument is naked. It infests the biliary ducts of sheep and cattle, but rarely affects man. The life-history of it is unknown.

DISTOMA HÆMATOBIUM.

In this fluke the sexes are distinct. The male is about half-an-inch long, has a flattened body, and towards the posterior part is grooved on the ventral surface to form a longitudinal canal for the female. The female is four-fifths-of-an-inch long, but much narrower than the male, and almost cylindrical in form. Both sexes have two ventral suckers, and the reproductive orifices are placed behind the posterior of these. The mature worm is a blood parasite, only found in the blood of the portal, hæmorrhoidal, mesenteric, and vesical veins. The ova pass through the walls of the bladder and colon, and in doing so set up inflammation and cause hæmorrhage, so that hæmaturia is almost a constant symptom of their presence. They chiefly affect man and the monkey, and are extremely common in Egypt, Abyssinia, the Cape, and Natal. In this country they are never seen except in patients who have brought them from Africa.

II.—CESTODA OR TAPEWORMS.

This group of parasites possesses the remarkable property of existing in two distinct forms, viz.,—as a colony of slightly different individuals, the so-called "tapeworm," and as a cystic parasite. The adult cestoid consists of a head with a number of segments or "proglottides." The head has suckers or hooklets, by means of which it can cling to the intestinal wall of its host. Each segment is sexually complete, and has both male and female reproductive organs. The head with its long chain of segments is termed a "strobilus." The segments multiply by gemmation from the head; the segments furthest away from the head end are therefore the more mature. These tapeworms have neither a mouth nor alimentary canal, but they possess a water vascular system.

TÆNIA MEDIOCANELLATA.

SYNONYMS—T. SAGINATA, T. INERMIS.

This is the commonest form of tapeworm seen in this country. It may reach the length of four yards, and have three or four thousand segments. The cystic form (*cysticercus bovis*) is seen in beef as small yellowish spots. The head is about one-eighth of an inch in diameter, is square, and flattened anteriorly. There are no hooklets or rostellum present, but at each corner of the head is a muscular sucker. From each of these suckers there runs a water vascular canal. There is a rounded opening in the centre of the head surrounded by a circular canal~~s~~, into which the four canals from the suckers open. From the central canal two longitudinal branches continue, one down each side of the various segments. The segments are longer than they are broad. The genital pore opens at the side of each segment, about the middle, and these pores are situated on alternate sides of the different segments. The uterus is relatively large, and branches dichotomously. In the anterior part of each segment is a convoluted tube; this is the testis, from which leads a duct ending in a penis. The small penis may sometimes be seen protruding through the genital pore.

TÆNIA SOLIUM.

This tape-worm is derived from pork, where the cystic form (*cysticercus cellulosæ*) is found. It occurs most frequently in Germany. It is a much narrower worm than the T. mediocanellata, and does not reach such a length. The head, which is not larger than the head of a small pin, has a well marked rostellum supporting a double row of hooklets—those of the lower row being the larger. The head is also furnished with four well-marked suckers. The segments are very much the same as in the former described parasite, but they are rather more slender. The uterus has seven to ten lateral branches, separated by intervals; each branch breaks up into a series of dentritic ramifications.

BOTHRIOCEPHALUS LATUS

Is the largest of all species of tænia, being fully larger than T. mediocanellata, and sometimes an inch in breadth. It chiefly occurs in Switzerland and in the north-west of Europe. Like the other two tapeworms above described, it lives in the small intestine of man. The body is thin and flattened like a ribbon. The joints near the head are very short and wide, looking like mere rings. From before backwards the worm becomes more and more slender, and at length appears like a mere thread. The segments near the tail-end are very narrow compared with those near the head, but are longer. The head is a simple structure, destitute of suckers, hooklets, and rostellum. It is about $\frac{1}{10}$ of an inch long by $\frac{1}{25}$ broad. It is club-shaped, and has a suctorial groove running down each lateral margin. About the middle of the ventral aspect in each segment is a small pigmented spot. In the centre of this spot is the genital pore. The pigment is due to the presence of dark coloured ova. The uterus lies in the centre of each segment, and consists of a rosette-shaped tube. The water vascular system is similar to that of other members of the group. Certain forms of fish are supposed to be the intermediate hosts of this parasite.

CYCLE OF DEVELOPMENT OF THESE FORMS.

During the act of defæcation, sexually matured segments or pro-

glottides are voided from the intestine. As these proglottides undergo disintegration, the ova are set free, and are carried into water, or some other medium, from which they are taken up by their intermediate host. The gastric juice of the intermediate host dissolves the ovum or shell, and the embryo (proscolex), by means of three pairs of hooklets, bores its way through the wall of the stomach or intestine, and reaches the tissue of its first host. When it settles in an organ the hooklets disappear and the proscolex undergoes metamorphoses, and at the end of two or three months is transformed into a vesicle filled with serum. From the inner surface of the wall of this vesicle there arises a new head or *scolex*. The scolex soon develops hooklets, sucking-discs (if present in the tapeworm form), and a water vascular system. During all this time the presence of this parasite, in the tissues of its host, is setting up local inflammatory changes, so that the creature becomes enclosed in a capsule of fibrous tissue. If now the intermediate host be used by man for food the cyst wall becomes dissolved by the gastric juice, the head attaches itself to the wall of the intestine, proglottides are developed behind it, and the full adult tapeworm is reached.

TÆNIA ECHINOCOCCUS (HYDATID CYST).

This parasite only occurs in man in the cystic form. The mature tapeworm infests the intestines of the dog and wolf. It is a worm about a quarter-of-an-inch long, and consists of four segments only. The first segment, or head, resembles that of T. Solium, but is smaller. It has a rostellum, which is surrounded by a double row of hooklets, some thirty or forty in number, also four suckers. The terminal segment, which is one-half of the whole length of the tapeworm, is the only one that comes to sexual maturity. It has a lateral genital pore placed a little behind its centre. The ovaries are complicated, and contain numerous ova. It has also a well developed water vascular system.

The cystic or immature form of this parasite occurs most frequently in the liver and peritoneum, to a less extent in the lungs, spleen, heart, kidneys, muscles, &c. The size of these cysts varies greatly. They may be merely the size of a pin's head, or they may grow even larger than the organ in which they are developed. The cysts may be single, or three or four may occur in one organ. Each cyst is always enclosed in a capsule of fibrous tissue, the so-called pseudo-cyst, which is produced by a local inflammatory change. The cyst wall proper is composed of two layers—*(a)* the *ectocyst*, or outer layer; *(b)* the *endocyst*, or inner layer. The ectocyst is a white, opaque, highly elastic cuticle, and when torn tends to curl inwards. **Microscopically** it is seen to be composed of a number of laminæ, each lamina presenting a peculiar transverse striation. The endocyst consists of granular matter, cells, muscle fibres, and a vascular system. The endocyst is called the "proligerous layer," for when the cyst reaches the size of a walnut this layer begins to develop a series of small vesicles (brood-capsules). "The wall of these is likewise twofold ; but the cuticular layer is within and the proligerous layer without. On these brood-capsules develop numbers of heads or scolices" (Ziegler).

Each scolex or echinococcus head is $\frac{1}{60}$ – $\frac{1}{100}$ of an inch long. It has a body and an anterior part called the head. The head (which is identical with the head of the little tapeworm in the dog) has a rostellum with hooklets and suckers. The body contains some highly refractive particles, and has a pedicle.

These hydatid cysts contain a watery fluid which gives the reaction of sodium chloride, but it contains no albumen. Floating in the fluid are usually a number of scolices.

Hydatid cysts most frequently occur in Iceland, Germany, and Australia ; in this country they are more rarely met with.

III.—NEMATODA OR THREADWORMS.

The nematoids are all slender, cylindrical, elongated worms, without segments or appendages. They are covered with a thick elastic cuticle. The mouth is placed at the anterior extremity, and the intestine is straight and extends from end to end of the body cavity. The genital organs and their orifices are on the ventral aspect. They are bisexual, and the females are usually the larger. Development is usually direct, any metamorphic changes being very slight.

TRICHINA SPIRALIS.

As in the case of the cestoids this worm occurs in two forms.

(1) The **adult form** is found in the intestine of man and some animals. It is a small, slender worm ; the male is about $\frac{1}{18}$ of an inch long, while the female is $\frac{1}{8}$. The body is rather thicker behind than in front, and it is slightly bent upon itself. The head is pointed, unarmed, with a central mouth leading directly into an alimentary tract, which terminates (in the case of the male) with the seminal duct, in a cloaca The male has a bilobed tail, the cloaca opening between the lobes. The penis consists of a V shaped spicule. The female is rather stouter than the male, and rounded posteriorly. The genital organs consist of a simple ovary, uterus, and vagina. In the case of the female the cloaca opens about the anterior third of the body. "The mode of reproduction is ovo-viviparous, the eggs being hatched within the body of the mother."

(2) The **immature** or **larval form** is found in the voluntary muscles of the same animals in which the adult worm occurs, but the pig is more frequently affected than any other animal. The muscles of the abdominal and thoracic walls, diaphragm, and front of the thighs are

the most common sites for them. The immature trichina is a very minute creature, about $\frac{1}{30}$ of an inch long. It has a digestive apparatus, and imperfectly developed sexual organs. It is usually found coiled up, and if it has persisted for some time it becomes encapsuled.

Life history of Trichina.—If a piece of muscle containing larval trichinæ reaches the stomach of an animal, the capsule (if present) is dissolved by the gastric juice, and the trichinæ are set free. In about two days they come to maturity in the intestine, when they proceed to pair. On the sixth or seventh day the females may be observed to contain perfectly formed free embryoes in their interior. A single worm may bring forth 1,000 to 1,300 young. These embryoes pass out through the genital orifice, and soon begin to burrow through the intestinal wall, and so reach the peritoneum, pericardium, and other surrounding tissues. But they usually do not rest permanently until they reach voluntary muscle. Others are carried by the lymph and blood to remote organs. They penetrate the sarcolemma of the muscle fibre, and reduce the contents to mere debris. In about fourteen days they reach the full larval size, and by a slow process of chronic inflammation gradually become encapsuled. At first a cyst is formed of a chitinous secretion of the animal, afterwards a wall of fibrous tissue appears. It may take as long as nine months to complete the process. The mature form lives but a short time in the intestine (5 to 8 weeks), while the larvæ may live till the death of the host. Calcareous salts usually become deposited within the cyst, which gives it a white, chalky appearance.

While migrating through the tissues and invading the muscles, the embryoes, if there be very many, set up a febrile condition which resembles typhoid fever. Differential diagnosis may be made by removing a small portion of muscle from the patient, and examining it carefully. The condition is known as *trichinosis*, and it sometimes ends in death. The parasites have been known to set up peritonitis. When they finally reach the muscles and become encysted, there is no danger to the patient.

FILARIA BANCROFTI (FILARIA SANGUINIS HOMINIS).

This parasite occurs chiefly in tropical countries—China, India, Egypt, and Brazil, etc.

(1) The **adult form** is found in the lymphatics, where it gives rise to obstruction, leading to lymphangiectasis, elephantiasis, and lymph scrotum. It is a long slender filiform animal about three inches in length. The head is somewhat club shaped, and from it there runs a simple alimentary canal which has an oval opening near the tail, the remainder of the body being occupied by reproductive organs.

(2) The **embryoes** are found in the blood and urine, and give rise to hæmaturia and chyluria. According to Manson they spread among

individuals by the agency of mosquitoes, which take up the filariæ with the blood they suck. While in the body of the mosquito the filariæ pass through an intermediate stage of development. They are then voided into water, from which they are again introduced into the human body. The young filariæ present remarkable diurnal variations. They are only found in the blood during night; but if the patient sits up all night and sleeps throughout the day, the converse condition holds.

DOCHMIUS DUODENALIS

Is a round worm about one-third-of-an-inch in length. The mouth is furnished with teeth, whereby it can plunge into the tissues of its host. On the ventral lip are four curved teeth, on the dorsal lip are two straight ones. It inhabits the duodenum and upper part of the jejunum. Sometimes large numbers of these creatures are found in one subject, when they cause great anæmia by sucking the blood from the mucous membrane. The disease is known as *Egyptian chlorosis*, and chiefly occurs in Italy, Egypt, Brazil, and the West Indies.

DRACUNCULUS MEDINENSIS (GUINEA WORM).

This is a very long slender thread-like worm, being about six feet in length, by $\frac{1}{10}$ of an inch in thickness. The body is uniformly cylindrical. The anterior end is rounded, the posterior terminates in a curved and abruptly tapering tail. In the rounded head is a simple central mouth leading to a narrow intestine; but there is no anus present. The uterus occupies nearly the whole body cavity. The embryo lives parasitically in an intermediate host, which is a small crustacean found in drinking water; thence they reach the human stomach. They develop to maturity under the skin, giving rise to cutaneous abscesses. The legs and feet are the most common situations where they are found. The male worm has not yet been discovered. This parasite is most frequently found in Africa and Asia.

TRICHOCEPHALUS DISPAR.

This, the "whipworm," though very commonly met with in Paris and Germany, is a rare parasite in this country. The anterior two-thirds of its body is very fine and thread-like, and by means of this delicate neck it anchors itself in the mucous membrane of the cæcum and upper part of the colon, but apparently gives rise to no symptoms. The male is about 1½ inches long, the female 2 inches. The posterior part of the body is thick and contains the genital organs, and the female genital orifice is at the junction of the thick and thin parts.

OXYURIS VERMICULARIS.

This worm is commonly met with in children, and gives rise to great itching and uneasiness. Their chief habitat is the large intestine, but they

are frequently found in the rectum. It is a small worm measuring $\frac{1}{6}$ of an inch in length; the female being about double that length. The body of the female is acuminate posteriorly: that of the male is blunter, and often bears a spine. They occur in vast numbers, and migrate from the rectum into surrounding parts, sometimes entering the vagina. It is probable that they are introduced into the alimentary tract through the drinking of contaminated water. As the eggs are not destroyed by being dried, it is easy to understand how they may be carried from place to place, and ultimately reach drinking water.

ASCARIS LUMBRICOIDES.

As its name implies, this parasite is very like the common earth worm in appearance. The female is from 10 to 14 inches long, and a $\frac{1}{4}$ of an inch thick. The male is 4 to 6 inches long, and more slender than the female. The male has a curved tail, on which are two sharp spines. The female tail is straight and possesses no spines. The female genital orifice is in the anterior half of the body. The ovaries consist of two tortuous tubes, which, when unravelled, measure about four feet long; they contain great numbers of eggs. The mouth is surrounded by three muscular lips, bearing very delicate teeth. Their habitat is the ileum, but they frequently migrate into the stomach and other parts. Often only one worm exists, but many more may be found in one patient. It is supposed that they enter the human body through the medium of drinking water as small embryoes, but their life history is not fully known.

Symptoms.—The presence of a single worm gives rise to no serious trouble. But when they are in large numbers they set up colic, constipation or diarrhœa, and vomiting. By obstructing the bile duct they may induce jaundice. Nervous irritation frequently results from their presence, and in children convulsions are common.

PART II.

SPECIAL PATHOLOGICAL ANATOMY.

DISEASES OF THE BLOOD.

The blood is not, properly speaking, a tissue, but we may speak of it as "a fluid tissue with unicellular elements." Most diseases of the blood are secondary conditions, but there are certain diseases in which we find morbid changes in the blood alone.

ANÆMIA.

We mean by this a diminution in the total amount of blood in the body (="Oligæmia"), or in certain of its constituents. In anæmia, so called, there is generally deficient quantity together with altered quality of the blood.

Chlorosis is a form of anæmia very common in young women, associated with a peculiar greenish yellow colour of the skin, and occurring without any obvious exciting cause.

Causes of anæmia.—I. Hæmorrhage. This may take place from wounds or rupture of blood-vessels. Should the bleeding be excessive, rapid death may follow. In such a case the death may be attributed to asthenia (loss of heart's action from imperfect blood supply), or to cerebral anæmia—the latter especially where death is very sudden. Death does certainly not result from loss of nutrition, because the patient can be kept alive for some time by simply injecting saline solutions to compensate for the loss of fluid. *Post mortem*, the skin, mucous membranes, and internal organs all show pallor, and the left heart is contracted and nearly empty. But in exceptional cases the lungs are œdematous and congested—an important point medico-legally. The total quantity of blood which may be lost without producing death is probably more than one-half (Greenfield). Anæmia may be produced slowly by repeated losses of blood from hæmorrhoids, or from

an ulcerated stomach. II. Discharges of a chronic nature, *e.g.*, suppuration and albuminuria in Bright's disease. III. Chronic starvation, as in cancer of the œsophagus. IV. Poisons.—(*a*) *From without.*—Certain poisons, especially lead and arsenic, when long administered cause impairment of the blood by diminishing certain of its constituents. (*b*) *From within.*—The specific virus of malaria no doubt vitiates the blood, for anæmia is one of the later symptoms of malarial disease. In cases of uræmic poisoning, a similar condition obtains, showing that urea in excess has a deteriorating influence on the blood. Rheumatic fever may be included in the same category.

In very many young women, anæmia, or the more aggravated form **chlorosis** arises without any very evident cause. But no doubt, the condition may be partly attributable to developmental agencies, *i.e.*, the tissues grow in a ratio proportionally faster than the blood-preparing glands can keep pace with. In a number of cases there has been discovered post mortem a condition of atresia of the aorta beyond the origin of the large vessels. Further, imperfect hygienic conditions undoubtedly exert a powerful exciting influence on the disease. Lack of fresh air is one of these conditions. It is a remarkable fact also that deprivation of the summer sunlight acts in the same direction. In the case of domestic servants, who often spend a large portion of the day in area flats, both of these conditions can with certainty be demonstrated to be at work. In these cases of so-called spontaneous anæmia, one finds a large diminution in the number of red blood corpuscles. The red cells are often increased in size, and they do not form rouleaux so readily as in normal conditions. The white corpuscles apparently do not participate in these morbid changes.

There is much doubt as to the exact **pathology** of chlorosis. It has been attributed to deficiency of iron in the blood ; to excessive destruction of blood ; to the absorption of poison or foul gases into the blood from the intestine, with consequent destruction of the red blood corpuscles. Others have theorized that chlorosis is the result of defective nerve action, occurring at the age of puberty, and interfering with the proper formation of the blood. Examination of the blood of a chlorotic girl shows that there is a great diminution of red cells. But it is important to note that the deficiency of hæmoglobin is relatively much greater, *i.e.*, the red cells that remain do not contain their full complement of colouring matter. This is an essential point of distinction between chlorosis and pernicious anæmia.

Reparation after loss of blood.—The water of the blood is quickly restored, probably by osmosis from the tissues. The solids are restored more slowly, the red cells regaining their full number later than the white. The former are evidently derived from the bone marrow and other blood glands.

PERNICIOUS ANÆMIA.

(SYNONYMS—"PROGRESSIVE," "IDIOPATHIC," OR "ESSENTIAL" ANÆMIA.)

This is a progressive, extreme, and very uncontrollable anæmia, developing without any evident cause. It often results in death. The disease was first pointed out by Addison, and it is characterized by a yellowish pallor of the skin, frequently along with hæmorrhages into the skin and retina. The main symptoms are palpitation, breathlessness, slight dropsy, increasing languor and weakness, and often some fever.

Post mortem, the bulk of the body is usually found unaltered, the quantity of fat being normal. But the fat may have an abnormal yellow colour. Hæmorrhages are found in various parts—the brain, subcutaneous tissues, mucous membranes, and serous membranes—in addition to those before mentioned. The liver is enlarged, firm, and has a peculiar brown colour. Fatty degeneration is found affecting the heart-muscle, walls of blood-vessels, and the epithelium of mucous surfaces. The heart also may exhibit brown atrophy. The hæmorrhages met with apparently result from the extreme fatty degeneration of the capillaries. The bone marrow is sometimes hypertrophied, and appears redder than normal ; microscopically, it may be found to contain cells with pigmented nuclei. Slight dropsies are frequently present in the subcutaneous tissues as well as in the serous sacs.

Condition of the blood.- There is an excessive diminution in the number of red blood corpuscles. They may sink to as low as 300,000 per cubic millimetre. (The normal number of red cells in the blood is about 5,000,000 per cubic millimetre in the male, and about 4,500,000 in the female.) Then they show great variation in size, ranging from perhaps three up to twelve micromillimetres. On the other hand, "microcytes," which are not normally present in the blood, are found in large numbers ; they are spherical bodies, about one-fourth the size of red cells, and of a deep yellow colour. Hæmoglobin is diminished in quantity, but to a relatively less extent than the cells. The richness in hæmoglobin is a noteworthy feature of the blood, as distinguished from the condition of chlorosis. The white corpuscles undergo but little change.

The **pathology** is obscure. The essential change seems to take place in the liver, as pointed out by Dr. William Hunter of Cambridge. There would seem to be an excessive destruction of red blood corpuscles, which takes place mostly in the portal system. The liver is extremely fatty, especially towards the centre of the lobules, while in the outer two zones of the lobules there is a very large quantity of red pigment, giving the reaction of iron. This iron is found to be present in amount many times above normal. But these conditions may only indicate exaggerated normal function on the part of the liver.

Aetiology.—The exciting causes of pernicious anæmia are quite unknown, but certain factors may be noted as influencing its occurrence: (1) Bad hygienic conditions. (2) Age and sex. The disease attacks women rather more frequently than men, although both sexes are nearly equally effected. It is a disease of middle life, differing in this respect from chlorosis; and in females it occurs at an earlier age than in males. (3) The disease is met with in particular districts.

LEUCOCYTOSIS

Is a condition in which there is increase in the number of leucocytes in the blood, apart from any other change. The white cells have been found increased as much as ten times in number.

The blood shows this pathological condition as a result of typhoid and other chronic fevers, and also of chronic suppuration such as occurs in cases of pyæmia.

LEUCOCYTHÆMIA.

This is a disease characterized chiefly by (1) a very great increase in the number of leucocytes in the blood, (2) a great diminution in the red corpuscles, and (3) an enlargement of the spleen. **Clinically**, it presents the following features:—the patient suffers from anæmia and slight fever, and there is a strong tendency to hæmorrhage in internal organs. Death generally results from hæmorrhage into the brain or from the mucous membranes. The constant tendency to hæmorrhage is shown by the fact that if the patient receives a slight wound, *e.g.*, through the extraction of a tooth, the bleeding is very persistent and difficult to arrest. The blood, as a rule, is thin, watery, and pale, sometimes turbid.

Classification.—Some authorities distinguish three forms of the disease, vizt.:—

I. Leucocythæmia Lienalis (Splenic).
II. Do. Lymphatica.
III. Do. Myelogenica.

According to Virchow, the difference between them consists in the fact that the excess of white corpuscles is formed in the spleen, lymphatic glands, and bone marrow respectively. Possibly, these forms may occur separately, but we commonly meet with combinations of two or of all three. They are said to be distinguished by the particular kind of leucocytes found in the blood.

Condition of the blood.—The appearance of the blood when drawn has been mentioned. Microscopically, we find an enormous increase in the leucocytes. At first sight they appear to be as numerous as the red cells, but in reality the proportion of white to red does not exceed 1 to 8. Then the leucocytes present variations in character, and usually several varieties are found in the blood of any single case. Thus we find (1) small non-nucleated cells, 4-5 m.m., (2) small nucleated cells, 7-8 m.m., (3) larger cells with three nuclei, 10-12 m.m., (4) the common large, coarsely granular, leucocyte. The first variety is said to be specially observed in the "myelogenic" form, the second and third in the "lymphatic" form, while the fourth is said to preponderate in the "splenic" form. But in addition, we sometimes find a large leucocyte having a horse-shoe shaped nucleus. All the leucocytes are more or less swollen and vesicular looking, and there is an absence of amœboid movement in them. They tend also to fatty degeneration, and altogether they come to resemble pus cells. With regard to the red cells, they are diminished in quantity, often to an extreme degree. Their size varies, and sometimes microcytes are seen amongst them. The bloodplates have been found to be very scanty, which probably accounts for the great hæmorrhagic tendency. The blood contains excess of hypoxanthin. When the blood has been kept for a time, needle shaped octohedral crystals are formed in it: these are known as "Charcot's crystals." They are soluble in dilute acids or alkalies, insoluble in ether, chloroform, or alcohol. Their composition is unknown, but they appear to be the phosphate of some alkaloid.

Morbid Anatomy.—Many morbid changes are found resulting from the altered state of the blood and diminished oxidation in the tissues. Small hæmorrhages are found scattered through the organs; sometimes large hæmorrhages are seen. The cause of these hæmorrhages is primarily embolic plugging of the vessels with leucocytes, which probably leads to their degeneration and consequent rupture. The **spleen** is uniformly enlarged, often weighing 7 or 8 lbs. The capsule is thickened and adherent to the abdominal wall, diaphragm, and liver. The surface shows dark or light patches, corresponding to recent and hæmorrhagic, or old and caseated infarctions. The organ is commonly firm. On section, the colour is of an even pinkish hue, the organ appears fatty, and feels greasy. Numerous infarctions may be seen, both old and recent. The pulp is dark and the Malpighian bodies stand out as paler spots. Microscopically, there appears to be hypertrophy of the pulp: the sinuses are distended, and contain proliferated endothelial cells and numbers of leucocytes, many of them larger than usual and showing traces of blood pigment in their interior. The Malpighian bodies exhibit a fibrous hyperplasia, often combined with a hyaline change that simulates waxy degeneration, and has been confounded with the latter. As the connective tissue increases, the cells become atrophied. The change is closely allied to that taking place in the lymphatic glands in Hodgkin's disease (Greenfield). The **lymphatic glands** are very rarely affected without the spleen. In many cases of splenic leucocythæmia, the lymphatic glands are enlarged. The **bone**

marrow is sometimes hypertrophied, its yellow colour changed to red, and crowded with leucocytes. Various organs, especially the **liver** and **kidneys**, show extensive infiltration with leucocytes.

Pathology.—Virchow theorized that the disease was due to excessive formation of leucocytes by the spleen. More recently, the disease has been said to be of microbic origin.

PLETHORA.

This means an increase in the quantity of blood above normal. But it is doubtful whether such a disease exists, because, in the first place, we don't know what limits there are to the normal quantity of blood in the human body. The proportion of the weight of the blood to that of the body is about 1 to 18 in a newly born child, and 1 to 14 in an adult. It has been ascertained by experiments on animals that if the total quantity of blood be raised to over a tenth of the body weight, the result is fatal on account of the great rise in the blood pressure. When the increase comes short of causing death, the effect is transient and soon passes off. The excessive fluid is drained away by the bodily secretions, while the superfluous red corpuscles are destroyed by the liver and other glands. **Transfusion** of blood in this way can be carried on with safety so long as we do not employ "dissimilar" blood, *i.e.*, blood of a different kind of animal. Otherwise, the process is attended with danger to the receiver. In the human subject, transfusion is often resorted to in cases of anæmia from various causes. The main object here aimed at is to give the heart sufficient fluid to act upon and so prevent its stopping. Injection of simple saline fluids is sometimes adopted for the same purpose.

EXCESSIVE QUANTITY OF PRODUCTS OF TISSUE METABOLISM IN THE BLOOD.

Uric acid accumulates in the blood in gouty conditions, as demonstrated by Garrod; and in cases of rheumatism *lactic acid* is found in excess. Christison showed that in Bright's disease *urea* accumulates in the blood. In diabetes mellitus *glucose* can be obtained from the blood. In other conditions *creatin* and *creatinin* can be separated.

ABNORMAL CONSTITUENTS IN BLOOD.

LIPÆMIA

Means an accumulation of *fatty globules* in the blood. Normally the blood contains a small amount of fat in a state of combination. In this condition free fat accumulates to such an extent as to give the blood a milky appearance. When drawn and allowed to stand, the fat droplets form a pale layer on the surface of the blood. The condition has been observed in diabetes mellitus; to a lesser degree in cancer. The fat is mostly found in the portal system of veins. How it occurs is unknown.

ACETONÆMIA.

The substance *acetone* (C_3H_6O) exists in traces as a normal constituent of the urine, in which it can be detected by the addition of a solution of perchloride of iron. In certain conditions, the substance is found in the blood, and the term "acetonæmia" is employed to indicate this phenomenon. "Diabetic coma" has been ascribed to the presence of acetone in the blood; and the patient's breath often possesses the peculiar odour of acetone. The condition is first manifested by the occurrence of "acetonuria," *i.e.*, excess of acetone in the urine. But acetonæmia is not a constant accompaniment of diabetes, nor, when present, does it always give rise to cerebral symptoms. Further, acetonæmia has been observed where there was no diabetes, thus, in pyrexial conditions, acetone has been found in the blood, as well as in excess in the urine.

MELANÆMIA ("BLACK BLOOD")

Is a name applied to a condition in which there is free pigment in the blood. It occurs after, sometimes during malarial fever, when the blood assumes a very dark colour. On microscopic examination, we find masses of blood pigment in the leucocytes and blood plasma. Osler and others have severally described certain constant appearances, evidently different stages in the development of a particular organism. Thus there have been described (1) a simple protoplasmic mass, floating free in the blood. At first it is crescentic shaped, afterwards oval, and in its centre there is a group of pigment particles. This enters a red blood corpuscle and becomes (2) a "plasmodium," which corresponds to the resting stage. This plasmodium appears as a granular mass, possessing independent movement. The granules are pigment, and are probably derived from the corpuscle, which becomes destroyed. The plasmodium then leaves the corpuscle, and appears next as (3) a ciliated organism, having a nucleus and two or three cilia. It is not so large as a red blood corpuscle. Probably it in turn is taken up by a leucocyte, and conveyed to the tissues.

Post mortem, deposits of pigment are found in various organs, including the spleen, liver, and bone marrow. The spleen is often abnormally dark in colour. The pigment is evidently carried to the different tissues by the activity of leucocytes.

DISEASES OF THE LYMPHATIC SYSTEM.

I. LYMPH VESSEL TRUNKS.

LYMPHANGITIS,

Or inflammation of lymph vessels, usually results from the absorption of irritant or septic matter from wounds. Very often a slight superficial abrasion on the hand becomes poisoned, and the lymph vessels under the skin are rendered visible in the form of pink lines extending up the arm. They are swollen and tender. The inflammation soon subsides as a rule, though sometimes points of suppuration form.

Morbid Anatomy.—The wall of the lymphatic is swollen and infiltrated, as well as surrounded with leucocytes. The endothelium swells and partly desquamates. The lymph may be coagulated, and appears fibrous.

If the lymphangitis becomes **suppurative**, the lymphatic wall undergoes inflammatory thickening, and the phenomena observed are exactly homologous with those in phlebitis (Greenfield). Abscesses form here and there, mostly in the lymphatic glands; thus in the case of a poisoned wound of the hand we might find them at the elbow.

In the disease *glanders* or *farcy*, the lymphatics show very similar changes. Under prolonged irritation they become corded, and tend to suppurate. In connection with this, small nodules, called "farcy buds," form in the ~~lungs~~. skin

The specific bacillus of *tuberculosis* probably infects first by way of the lymphatics, hence the reason why lymphatic glands are so early involved. It sets up inflammatory changes in the vessels very much like those described, and leads to softening.

LYMPHANGIECTASIS,

Or dilatation of lymphatic trunks, is usually the result of mechanical obstruction. Since the anastomosis between lymphatic vessels is very free, it is evident that this is not a common disease.

Effects.—I. Varicose dilatation of the lymphatics affected. This is seen sometimes in the superficial lymphatics, *e.g.*, of the lower limb. The vessels are swollen, and present beaded dilatations which correspond to the valves. These dilatations may ulcerate or rupture from accidental damage, and give rise to "lymphatic ulcers" and "lymphorrhagia." II. Chronic dilatation of lymph spaces. This in an extreme degree gives rise to the condition **elephantiasis**, where there is great swelling of part or of the whole body. The obstruction here is caused by the organism *filaria sanguinis hominis*, the ova of which become impacted in the glands (*v.* page 81). Sometimes the scrotum is alone affected, and such a condition is called **lymph scrotum.** Occasionally rupture of the urinary tract takes place, and the lymph is discharged into the urine, constituting **chyluria.** The condition known as **molluscum fibrosum** may result from this lymphangiectasis, and it is also the cause of **macroglossia.**

NEW GROWTHS.

Various forms of new growth may affect the lymphatics, especially cancer and tubercle. But it must be borne in mind that cancerous and other material may produce no irritation in the lymphatic vessels along which it is conveyed, and only attack the glands through which it is filtered.

II. LYMPHATIC GLANDS.

ATROPHY

Is very commonly seen in old age. The atrophy may be associated with overgrowth of the fibrous stroma, thus constituting a "fibrous atrophy." It is sometimes accompanied by **fatty degeneration.**

WAXY DEGENERATION

Of lymph glands is very often found along with waxy degeneration elsewhere.

Naked eye char.—The gland is smaller. On section it is smooth and may show small glossy patches, which are rendered more evident with iodine staining.

Micro. char.—If seen early, the only parts affected are the ultimate arterioles and their immediate capillary branches. Later, the capillary plexuses in the follicles become waxy, and lastly the connective tissue fibrils of the stroma. Rarely, the endothelial cells may become affected in advanced cases. Sometimes the lymph corpuscles atrophy, and then the supporting framework of the gland can be seen to advantage.

PIGMENTATION

Of lymph glands with extraneous pigment is a common occurrence. A typical instance of it is seen in the bronchial glands as the result of inhaling carbon particles; in coal miners this becomes extreme. The carbon passes from the air cells into the different lymphatic vessels, and is carried by the lymph stream toward the glands. It is primarily deposited in the lymphatics round the gland, later in the gland itself. Under the microscope, the black particles are seen to occupy the sinuses of the gland mainly, and are contained in branch ed corpuscles. By and by the pigmentation affects the entire gland.

Effect.—Pigment particles in a gland tend to cause irritation,

leading to proliferation of the endothelium of the lymph sinus, followed by increase in the connective tissue, and consequent induration of the gland.

LYMPHADENITIS.

Or inflammation of lymph glands is extremely common. The condition may be acute or chronic.

A. ACUTE.

Cause.—Acute lymphadenitis results from the introduction of various irritants, mostly specific. Thus in diptheria, the glands about the neck and jaw become inflamed, and the specific poison of typhoid fever sets up inflammation of the mesenteric glands; then a poisoned wound of the foot is frequently followed by the formation of a bubo in the groin.

Morbid Anatomy.—The gland swells rapidly, and becomes deeply mottled with a red injection. There may be small or diffuse hæmorrhages, if the irritation is great. But in a very short time, the gland shows evidence of reaction and becomes paler than normal—a fact we would not be led to expect. When a number of neighbouring glands are inflamed, the inter-glandular tissues may become involved and show inflammatory œdema. **Microscopically**, the gland is seen to be crowded with leucocytes. Numbers of degenerated red blood corpuscles are found in the lymph sinuses and swollen trabecular vessels. Fragments of endothelial cells are also found in the lymph, having been derived from proliferation of the lining of the trabeculæ. The lymph is coagulated in the sinuses. If one regards the gland as a convoluted piece of omentum, the changes observed in the latter during inflammation are practically identical with those occurring in the gland (Greenfield.)

Results.—(1) Return to normal. (2) If the inflammation is intense, points of suppuration arise, become confluent, then break down and form an abscess. This may either discharge or dry up, and lead to caseation and subsequent calcification. (3) Prolonged inflammation may become chronic, resulting in hyperplasia and induration, with thickening of the capsule.

B. CHRONIC.

A simple chronic lymphadenitis frequently results from prolonged irritation of the glands, *e.g.*, from the introduction of foreign substances, where the solid particles are conveyed to the gland. Thus we saw that pigment particles may lead to enlargement and often induration of the glands in which they are deposited. Further, a chronic inflammatory process may be said to occur in "Hodgkin's disease," and in tuberculous disease of lymph glands.

TUBERCULOSIS

In lymph glands is usually of a chronic nature. It is often a primary affection, as instanced in the very common occurrence of enlarged "scrofulous" glands in the necks of children. It also occurs secondarily by extension from infected organs. Thus, in phthisis the bronchial glands frequently become tubercular, likewise the mesenteric glands in connection with tuberculosis of the intestines. But we occasionally find enlargement of the mesenteric glands without any evident tuberculosis of the intestines or other parts.

Varieties.—I. **Chronic Fibroid Tubercle.**—In long continued phthisis, the bronchial glands become enlarged and firm ; and on section they show scattered grey granulations, which occasionally turn caseous. Microscopically, these are shown to consist of typical giant cell systems, which have become fibrous, and appear to be continuous with the stroma of the gland.

II. **Large-celled Hyperplasia.**—Here, while we may find a few giant cell systems, the characteristic appearance presented under the microscope is a swelling and proliferation of the endothelium of the gland. It is comparable to the change that takes place in the pulmonary air cells in acute tubercle. The process often results in rapid caseation, beginning at a number of different centres simultaneously.

III. **Acute Tuberculosis.**—In this condition the glands become greatly enlarged and fuse into masses by periadenitis. Complete caseation follows, and the microscope reveals little but an amorphous mass, the capsule alone showing traces of inflammation. The structure of the gland is lost, but possibly a giant cell may be distinguished here and there. If we were to look early enough, we should find enormous numbers of tubercle bacilli in and around the gland, but these have disappeared in the advanced stage of complete caseation. A section of the gland in such a condition has been compared to that of a raw potato, from its yellow colour and firm consistence. In course of time it may either suppurate or become gradually infiltrated with lime salts, then dry up and become hard as stone.

SYPHILIS

Often causes enlargement of glands, *e.g.*, a chancre gives rise to swelling of the glands in the groin.

Char.—The glands are enlarged and indurated, and appear grey and non-caseous on section. Microscopically, the chief change is a proliferation of connective tissue in the gland. No characteristic cells are found.

HODGKIN'S DISEASE (OR LYMPHADENOMA)

Is a progressive enlargement of lymph glands, commencing in the neck, then extending to the axilla and gradually over the body ; associated

with anæmia, emaciation, some irregular fever, and very often enlargement of the spleen, as well as secondary lymphoid growths in the spleen and other organs.

Morbid anatomy.—If we examine a mass of glands in the neck, we find them enormously swollen and matted together by firm fibrous tissue. On section they are firm, commonly greyer than natural, and pigmented, with perhaps some caseation. **Microscopically,** the essential change is a proliferation of endothelium and a hyperplasia of the supporting connective tissue, the latter no doubt due to the irritative overgrowth of the endothelium. Gradually the connective tissue comes to preponderate in the gland, and the lymph cells practically disappear. In the **lungs, liver,** and **kidneys,** the secondary growths closely resemble the enlarged lymphatic glands. The condition of the **spleen** will be referred to in studying the diseases of that organ. As regards the **blood,** the red corpuscles are diminished in number (accounting for the anæmia), but there is no increase in the number of leucocytes. The disease was formerly called "lymphatic leukæmia," but the essential feature in leukæmia is wanting in this disease. The only excuse for the name is the enlargement of the spleen that occurs in both diseases.

TUMOURS.

I. **Primary.**—**Lymphoma, lymph-adenoma,** and **lympho-sarcoma** are the commonest. They resemble each other, and besides are not easily distinguished from inflammatory enlargements. They occur oftenest in the mediastinum. Ordinary round or spindle celled **sarcoma** is sometimes found in the retro-peritoneal glands near the head of the pancreas, and may grow to a large size.

II. **Secondary.**—Lymph glands are liable to be the seat of all forms of secondary growth. **Cancer** in its various forms is very common. Secondary **sarcomas** are seldom found, however.

DISEASES OF THE SPLEEN.

General considerations.—The spleen is an organ not essential to life. It has been removed without injuriously affecting the health. Its size does not bear a constant proportion to that of the body ; it is very erratic in this respect. Frequently, there are found small accessory spleens (splenicnli), which lie near the main organ, and are subject to the same morbid influences.

PERISPLENITIS,

Or inflammation of the capsule, often causes thickenings and adhesion to surrounding parts. These are chiefly seen in the convex surface, and give rise to white plate-like masses, having a simple laminated structure.

They are commonly spoken of as "flat fibromas." Such thickenings do not implicate the substance of the organ, though in syphilitic subjects we find an extension of fibrous bands into its interior.

ATROPHY

Of the spleen occurs constantly as a senile change, the organ becoming very much smaller.

FATTY DEGENERATION

Of the spleen has chiefly been observed in cases of prolonged fever; but little is known about it.

HYALINE DEGENERATION

Is typically seen in the spleen. The exciting causes are fevers, particularly scarlet fever and diptheria. The changes in the arteries have before been studied.

WAXY DEGENERATION

Is the commonest disease of the spleen, and may affect that organ alone. If there is waxy degeneration elsewhere, the spleen is always affected (Greenfield). There are two forms :—

I. SAGO WAXY.

Naked eye char.—The organ is somewhat enlarged, and retains its normal shape. It is firmer than natural. The general colour—on the surface as well as on section—is dark, an exceptional circumstance in waxy organs. This is caused by excessive dilatation of the venous sinuses and replacement of the lymph by blood. Thus, it is not a true congestion. On section, the organ is seen to be studded with small rounded points, resembling grains of boiled sago—gelatinous and translucent—occupying the position of the Malpighian corpuscles. They are distinguished from tubercle granulations by the fact that they are not prominent above the level of the section. Iodine stain gives a mahogany brown reaction with the waxy points, while the surrounding healthy tissue becomes yellow.

Micro. char.—The parts affected are almost exclusively the Malpighian corpuscles, and particularly those in relation to the more distal portions of the arterial tufts or pencils. In an early stage, the waxy change is limited to the capillary plexus, next the connective tissue filaments of the adenoid sheaths are attacked, and then the central arteriole becomes involved. The adenoid cells remain healthy, unless in very advanced cases, when possibly they also are affected. At any rate, they are not readily discerned; but that may be accounted for by their having become atrophied from pressure (Woodhead). In addition to these appearances, careful examination reveals scattered patches and lines of waxy tissue in the splenic pulp, corresponding to small arterioles.

II. DIFFUSE WAXY.

Naked eye char.—The spleen is greatly enlarged, its edges are thickened, and it has a firm consistence like bacon. The colour is not so dark as in the other form. On section, the margins are seen to remain sharp, and the surface has a peculiar glistening appearance, common to all waxy organs. On treating with iodine fluid, an extensive brown colouration is got, the Malpighian bodies very often remaining unstained.

Micro. char.—It will be observed that the disease affects the walls of the venous sinuses in the splenic pulp, producing a honeycombed appearance under the low power of the microscope. The Malpighian bodies show little or no change. But sometimes, the Malpighian bodies are implicated with the pulp, and in those cases the condition is probably an advanced stage of sago waxy.

PIGMENTATION

Of the spleen, in the form of small coloured patches, is often observed in the case of individuals who have suffered from malaria. Very rarely, deposits of pigment are found in other conditions.

VENOUS CONGESTION

Of the spleen occurs normally during digestion, the organ undergoing enlargement to compensate for the dilatation of the portal system, thus acting as a "portal heart." Pathologically, it occurs whenever there is obstruction to the portal circulation, so it is commonly a chronic condition, and results from disease of the liver, heart, or lungs.

Characters.—The organ is more or less enlarged, usually in all directions, though sometimes it is specially thick. It is firm in consistence, and dark in colour. The capsule is often thickened. On section, the edges remain sharp, the surface of the section being uniformly dark. The Malpighian corpuscles are usually invisible. **Microscopically,** the venous sinuses are greatly distended, the result being that the other tissues of the pulp are atrophied from pressure. The trabeculæ are often thickened, and pigment from altered blood may be deposited.

EMBOLISM AND INFARCTION.

Embolic infarction of the spleen is extremely common. Possibly the large size of the splenic artery may account for this. The fact that its branches are all "end arteries" renders them very liable to the consequences of infarction. Heart disease is a very common cause of the condition.

Characters.- As a rule we do not find solitary infarcts; it is more usual to get several in various stages of existence. They tend to run across the organ on its convex surface, and are readily distinguished by their colour and firmness from the general splenic substance. On cutting into one of them it is seen to be wedge-shaped, the base of the wedge being at the surface, the apex directed towards the hilum. It is surrounded by a zone of congestion. If the infarct be quite recent, it will be of a deep red colour. Gradually, however, it becomes paler—this pallor spreading from the centre outwards—until the whole infarct is of a yellowish-white colour, with the exception of a more persistent zone of congestion at the periphery. Ultimately this zone is replaced by a capsule of fibrous tissue, while the pale interior undergoes fatty degeneration. Later, the fatty material becomes more or less absorbed, and the capsule simultaneously contracts, until in time nothing remains but a scar on the surface of the organ. This would seem to produce little or no injurious after-effect on the spleen. But this process of simple cicatrization does not always obtain. Occasionally, a local peritonitis is set up over the infarct, fibrin is deposited, and adhesion follows ; or, rarely, it leads to a more diffuse peritonitis. Then, again, infarcts sometimes suppurate and form abscesses, but in these cases a septic condition probably exists. When rapid multiple embolism occurs in the spleen, we find that the organ enlarges as a whole, the obstruction of the arteries leading to a collateral hyperæmia by distension of those remaining patent (Coats).

ACTIVE HYPERÆMIA AND INFLAMMATION.

The spleen undergoes inflammatory enlargement in many acute general diseases, particularly fevers of various kinds and syphilis. The name of **acute splenic tumour** is sometimes applied to this condition ; and no doubt the irritating state of the blood in fevers is an exciting cause of the inflammation. In this country we see it most marked in cases of typhus fever, and it also occurs to an extreme degree in the malarial fevers of tropical climates.

Morbid anatomy.—Suppose we examine the spleen in a case of fatal **typhus.** It is found much enlarged—causing stretching of the capsule—softened (perhaps diffluent), and very vascular. On section, the colour is dark red, becoming greyish-red in prolonged cases, and greasy looking from fatty changes. On scraping the surface then, a dark pus-like fluid exudes. Microscopically, the blood-vessels are found gorged with blood : in an early stage this is most marked near the margins of the adenoid sheaths of the vessels ; later, it becomes general. The pulp contains an excess of leucocytes, and the epithelium lining the trabeculæ swells up. Now, were we to examine the spleen in a case of **typhoid, scarlet, smallpox,** or **diptheria,** we should find somewhat different appearances. In these diseases, the organ, while it may be considerably enlarged, is firm, instead of being soft : and on section, the Malpighian bodies are seen to be swollen and prominent. Under the microscope, the adenoid tissue shows pronounced increase. Again, in

cases of **intermittent malarial fever**, the spleen shows certain special characters. In the first place, the organ can often be observed to swell during an "interval," and subside during an "intermission." When the patient dies after repeated attacks, the organ is found enormously enlarged, and of firm consistence, the term "ague-cake" being commonly applied to it. The capsule is thickened, and frequently shows patches of adhesions from perisplenitis. Microscopically, the connective tissue element is greatly hypertrophied, and there is a general hyperplasia of all the elements. In addition, pigment is deposited.

Thus, it will be evident that the appearance of the spleen may often lead us to suspect the nature of the disease from which the patient has died The only endemic fever in which the spleen does not become enlarged is yellow fever, and this is probably accounted for by the hæmorrhage from the stomach (Greenfield).

Results.—(1.) As a rule, with the subsidence of the disease the spleen diminishes in size, and returns nearly to its normal condition, though it may remain soft, with a wrinkled capsule. (2.) Intense fevers, such as typhus and malaria, sometimes cause such a rapid swelling of the spleen as to rupture the capsule. (3.) Suppuration occasionally results, usually in typhoid or malaria. When an abscess forms, it is commonly upon the surface, and, as a rule, it causes death by setting up fatal peritonitis. But an abscess has been known to make its way through the diaphragm into the pleura, or even to open posteriorly. (4.) The spleen may remain permanently enlarged, as we have seen in the case of the "ague-cake" in malarial fever. (5.) A perisplenic abscess is sometimes got in cases of inflammatory enlargement of the spleen, though it may result from other diseases of that organ. How it starts is unknown, but it is usually situated outside the convex surface, and limited by adhesions formed with the diaphragm or other contiguous part.

HODGKIN'S DISEASE

Produces striking changes in the spleen. The organ is greatly enlarged, but retains its general configuration. In consistence it is firm and fleshy, and its colour is dark from congestion, though numerous purple patches can also be made out on the surface. On section, the colour is deep red, and scattered through the pulp are numbers of small angular (perhaps rounded) translucent yellow masses, that have been compared to boiled suet. In a favourable section, they can be seen branching, evidently corresponding with the branching arteries. These masses are really altered Malpighian bodies, and they have undergone a process precisely analagous to that which we studied in the lymph glands. The **microscope** shows that there has been a fibrous hyperplasia of the adenoid sheaths, the stroma becoming denser at the expense of the lymphoid cells which gradually diminish in number. Added to that, there is a deposit of golden brown pigment around the advancing mass, derived from red blood corpuscles which have been assimilated by the cells lining the pulp. Perhaps this may have some connection with the anæmia, which is one of the features of Hodgkin's disease.

TUBERCULOSIS

Does not affect the spleen as a primary disease, but the organ is very commonly involved in general acute tuberculosis. It occurs in one of two forms :—I. **Acute Miliary Tuberculosis,** in which small grey granulations are often abundantly found near the surface, and in the capsule. On section, they are grey and translucent, and prominent above the surface ; frequently they show central yellow points, indicating caseation. The organ is enlarged and firm. This form is commonly observed in children dying of acute tuberculosis. II. **Ape Tuberculosis**.—This is uncommon in man, but it is sometimes found in children. The spleen may be either larger or smaller than normal. On section, the pulp shows congestion, and distributed through it we find large masses of tubercle, usually caseous, varying in size from that of a pea to a walnut. They resemble the suet like masses in lymphadenoma, but their yellow appearance usually suffices to distinguish them from the latter. The organ sometimes receives the name of "hardbake spleen." Microscopically, the ordinary characters of caseous tubercle are to be made out.

SYPHILIS

Rarely affects the spleen, though the effects may be manifested in different ways. In the first place, the organ has been found in a state of **hypertrophy** in cases of fatal congenital syphilis in infants. All the elements of the gland seem to undergo hyperplastic increase. Secondly, gummata have occasionally been seen, very similar to those in the liver *(q.v.)*. And, lastly, cicatrices and thickenings of the capsule have been observed.

PARASITES.

I. **Hydatids** are rare in the spleen. They resemble those in the liver. II. **Pentastoma denticulatum** occasionally produces small cysts, which usually caseate. As a rule they are near the surface of the organ.

TUMOURS.

I. **Primary.**—The commonest primary tumour is **sarcoma**. **Fibroma** and **angioma** have been observed.

II. **Secondary.**—**Sarcomas** occur secondarily in the spleen with greater frequency than any primary tumour (Coats). **Cancer** occasionally occurs.

DISEASES OF SUPRARENAL BODIES.

MALFORMATION

Of the suprarenals is very rarely observed. In case of absence of one kidney, the suprarenal remains. But the latter may be absent, and when such is the case malformations are generally present in other viscera. Accessory suprarenals have sometimes been found scattered in the region of the solar plexus.

PIGMENTATION

Occurs very constantly in old age, affecting chiefly the cortex in its deeper layers. Granules of pigment are deposited in the cells, which may become of a uniform yellow tint.

HÆMORRHAGE

Within the suprarenals is uncommon. Traumatic injury sometimes gives rise to enormous swelling of the organ. Spontaneous hæmorrhages occur in the course of scarlet and other fevers, and in pernicious anæmia (when we find them in nearly all organs). They are usually small and scattered.

TUBERCULOSIS

Of the suprarenals occurs in cases of acute general tuberculosis. It is manifested by the presence of scattered tubercular granulations.

MELASMA SUPRARENALE.

(ADDISON'S DISEASE).

This is a disease characterized by a bronzing of the skin, attended with symptoms of anæmia, vomiting, weakness and cachexia, and usually ending in death. The pigmentation commences in the more exposed parts of the skin, then affects parts where pigment is normally found (around the nipples, at flexures, etc.), and gradually extends until the whole skin shows a deep brown colour with a pigmented mottling. The mucous membranes are also affected ; the inside of the mouth appears as if it had been stained with black ink.

Post mortem.—The suprarenal bodies show more or less advanced changes. In an early stage, they are found enlarged, nodulated and adherent to their surroundings. On section, fibrous patches are seen, usually with a caseous centre, sometimes with an abscess or cyst formation. In an advanced stage, the entire suprarenal is converted into a caseous mass, surrounded by a thick fibrous capsule. Both organs are commonly affected. Under the microscope, the process turns out to be tubercular in nature : the general appearance is that of tuberculosis, and giant cells, as well as tubercle bacilli, can be distinguished. So the disease is really a local tuberculosis, though in a large number of cases there is tuberculosis in other parts. In many cases, changes have been observed in the abdominal sympathetic, and semilunar ganglia.

TUMOURS

Seldom affect the suprarenals. The one oftenest observed is that described by Virchow as **struma lipomatosa suprarenalis** which is a nodular growth consisting essentially of fatty glandular tissue (Ziegler). **Primary cancers** and **sarcomas** have occasionally been seen, and they may form very large tumours. Small **secondary cancers** also occur.

DISEASES OF THE THYROID GLAND.

MALFORMATIONS.

The thyroid is rarely absent as a whole, but portions of it are occasionally wanting. Sometimes it is abnormally large, and the lobes may be multiple. Small accessory glands have been found occupying various situations more or less removed from the normal thyroid body: they have even been discovered in the interior of the larynx. Cases of extreme rarity have been recorded, where the isthmus of the thyroid instead of lying in front of the trachea passed behind it (between the trachea and œsophagus).

ATROPHY.

In the disease *myxœdema*, which is characterized by œdema and mucoid degeneration of the tissues, with slowly increasing loss of mental power, we very constantly find a wasting of the thyroid gland. And it has been shown by experiment on monkeys that entire removal of the thyroid gland is followed by very similar nutritive changes. The thyroid atrophies normally in old age, like many other organs.

INFLAMMATION

Of the thyroid has been set up by injury, and it sometimes results from septic fevers. The inflammation may be so intense as to lead to suppuration and abscess formation. Abscesses sometimes rupture into surrounding parts. Acute inflammation attacks goîtrous as well as normal thyroids.

GOÎTRE OR BRONCHOCELE.

These are terms applied to many forms of general or localized enlargement of the thyroid gland. The enlargement may be congenital or acquired. A convenient clinical **classification** of the varieties of goître is into, I. Vascular, II. Cystic, III. Fibrous, IV. Exophthalmic.

I. **Vascular goître** usually occurs late in life and is a transient condition. It arises from a simple hyperæmia, causing an undue distension of the blood vessels. The enlargement is nearly always symmetrical.

II. **Cystic goître** is the commonest form, often causing enormous swelling of the gland. On examination the gland is found to be full of small cysts, which seem to result from hypertrophy of the gland follicles. The cysts usually contain a clear coffee-ground coloured fluid, sometimes tinged with blood. At other times, the contents are chiefly of a colloid nature, evidently due to increase of the normal colloid contents. A cystic goître is frequently a-symmetrical.

III. **Fibrous goître.** This results from a chronic hyperplasia of the fibrous stroma of the gland ; the follicles being obliterated and the gland becoming solid and indurated.

IV. **Exophthalmic goître,** or Graves' disease, is a condition in which the leading phenomena are (1) vascular disturbance, manifested by *(a)* rapidity and irregularity of heart's action, and *(b)* dilatation of vessels in the head, neck, thorax, and upper arms : (2) goître,—second in point of time. It is small in size, and is mainly of the "vascular" type, although there is commonly some hypertrophy of the gland texture. The occurrence of goître may be secondary or accidental in the disease : (3) protrusion of the eye-balls, "exophthalmos." This is the last to make its appearance, and is not always developed. When present, the individual cannot close his eyelids, and the eye is liable to become inflamed from the constant and undue exposure.

Aetiology of goître. At present, pathologists are not agreed as to the primary cause or causes of goître. No doubt, many cases can be explained by an increased blood supply to the organ, though how this comes about is unknown. But goître is essentially an endemic disease ; it is extremely common in the Alps and other mountainous districts, and in Derbyshire it is met with so frequently as to give rise to the expression "Derbyshire neck." Further, it is known that strangers taking up their abode in such districts often acquire the disease, whereas goîtrous individuals have been cured by removing from these districts into others where it is unknown. So, evidently, there must be some local agency—whether in the soil, the water, the air, or other medium—that favours the condition. Probably this local cause is of the nature of a miasma, arising from certain kinds of rock or soil. The mineral *dolomite* is largely found in many goîtrous districts. On the other hand, the exciting agent may enter the body in drinking water. But how it operates we cannot tell. We can gather that it has the power of influencing the unborn fœtus, because the disease is often congenital.

The condition of **exopthalmic goître** requires a further explanation. It appears to be of neurotic origin, involving the vaso-motor centre and perhaps the pneumogastric nerve. The entire vagus certainly cannot be implicated, because the lungs and stomach are not deranged; there seems to be a combined effect between some portion of the nerve with the vaso-motor centre. We know that the heart is very readily affected by the emotions, palpitating on excitement of various kinds; and possibly exopthalmic goître may follow a powerful shock applied to the nerve centre that governs the heart through the pneumogastric nerve.

The **function of the thyroid** has been the subject of prolonged investigation. It seems certain that the gland manufactures something which, absorbed into the blood, ministers to the nutrition of the nervous system.

TUMOURS.

Various forms of **adenoma** have been described. Wölfler speaks of (1) Fœtal, (2) Gelatinous, (3) Myxomatous, and (4) Columnar-celled. They are innocent tumours. The first-named develops from some embryonic residue, though its growth may not be apparent till puberty or pregnancy. It produces variously sized vascular nodes, having an imperfectly developed gland structure. The **gelatinous** form is really a cystic formation, the cysts being filled with colloid material. It constitutes one variety of cystic goître. The **myxomatous** adenoma results from a transformation of either of the first two forms. **Columnar celled** adenoma is a rare form. The tumour is made up of vesicles lined with columnar cells and traversed by irregular bands made up of the same kind of cells. **Malignant Adenoma** of the thyroid is a tumour that closely resembles carcinoma. It grows rapidly, is very vascular, and recurs after excision.

Carcinoma of the thyroid occurs in regions where goître is endemic, and usually develops in an existing goîtrous growth. It assumes various forms, but usually appears as a soft tumour situated in one lobe only, and having an alveolar structure. **Sarcomas** sometimes develop in the thyroid. They seldom involve the whole gland.

DISEASES OF THE THYMUS GLAND.

The thymus is a ductless gland, situated behind the manubrium sterni, and consisting of two elongated lobes attached along their contiguous borders. In structure it resembles a lymphatic gland, and there are numerous lymphatic vessels in relation to it. The thymus attains its full size in the second year of infancy, after which it remains unaltered for some years. About the tenth year it atrophies, and soon nothing remains to mark its site but some fibrous and fatty tissue with the remains of a few blood-vessels. Occasionally the gland persists in the adult for a number of years. This is the most common morbid change in connection with it, but it occasions no functional disorder.

INFLAMMATION

Of the thymus gland has been observed. Pyæmia has been known to cause pus formation in the gland. Secondary inflammation may be communicated from suppurating cervical glands in children.

TUMOURS.

In cases of *Hodgkin's disease* in children the thymus becomes the seat of a lymphadenomatous growth, similar to that which besets the lymphatic glands. Then, in *leucocythæmia*, sarcomatous tumours occasionally develop in the glands. Besides, the thymus forms the starting point for a number of mediastinal tumours, some malignant and others not (Greenfield). Their structure resembles that of a lymphoma very often.

DISEASES OF THE CIRCULATORY SYSTEM.

I. The HEART and its MEMBRANES.

The **muscle substance** of the heart, the **pericardium**, and the **endocardium** are liable to be commonly implicated in various morbid processes. But we shall take up in order the diseases which affect them severally, and refer incidentally to any joint condition.

A. The PERICARDIUM.

MALFORMATIONS.

These are few in number and uncommon. Total **absence** of the sac has been observed. The heart in such a case occupies the left pleural cavity. In other cases there has been partial deficiency. Occasionally, cyst-like diverticula have been seen.

HYDROPERICARDIUM

Is a name applied to the distension of the sac with dropsical fluid, *i.e.*, it is a dropsy of the pericardium. It is seldom seen alone, nearly always along with general dropsy. It gives rise to no special appearances, although when long persistent, the epicardial epithelium swells and becomes clouded. The amount of fluid in the sac is never great, unless there has been concurrent pericarditis.

MILK SPOTS,

So called, are very common pathological conditions, occuring in about half the cases examined post mortem (Coats). They are white opaque patches of thickened pericardium, situated on the anterior surface of the heart—usually over the right ventricle, less frequently over the apex of the left ventricle. They involve the visceral layer, less commonly the parietal layer of the membrane. These patches vary much in size, ranging from minute spots to areas almost as large as the heart itself, and they have no definite outline. They seldom reach any great thickness, but some of them grow so thick as to acquire a cartilagenous consistence. The **microscope** shows that the patches consist simply of thickened pericardium, with an epithelial covering.

Cause.—Milk spots were very common in soldiers in the days when "cross belts" were used to support their knapsacks. Accordingly, the name of "soldiers' spots" was formerly applied to them, and they were regarded as the result of friction from the heavy pressure of the belt over the præcordia. They are no doubt caused by a persistent irritation of the pericardium from contact with the sternum, which irritation sets up chronic inflammation in the membrane, and leads to its thickening. So the condition is constantly observed in cases of cardiac hypertrophy.

HÆMORRHAGES

Of very varying extent have been met with both in and within the pericardium. We may divide them into—

I. **Ecchymoses.**—Minute extravasations of blood take place into the pericardium (as into other serous as well as synovial membranes) in the course of many fevers and in some forms of blood poisoning. They have also been discovered in cases of death from obstruction to the cardiac circulation. The ecchymoses, as a rule, are limited to the epicardium in its deeper layers, not coming to the surface, and they are oftenest seen at the base of the heart, on its posterior aspect.

II. **Larger Hæmorrhages**, making free vent into the sac of the pericardium, may result from a number of causes. Thus, we may enumerate *(a)* traumatism, *e.g.*, a stab or other injury; *(b)* aneurism of heart or ascending aorta, leading to rupture; *(c)* pericarditis, either simple, tubercular, or cancerous.

PERICARDITIS,

Or inflammation of the pericardium, is a common enough condition.

The **causes** of it are numerous, but by far the most frequent of these is acute rheumatism. That disease furnishes at least 90 per cent. of the cases (Greenfield). Next in frequency is Bright's disease, and much more rarely it is due to specific fevers. Then, the inflammation may be secondary to inflammations of adjacent parts, vizt. :—the lung, with its pleural sac, the heart wall, and the mediastinum. Lastly, various growths in the membrane itself—including tubercle, cancer, and sarcoma—may set up inflammation. Very rarely indeed does pericarditis follow a traumatic injury.

Characters, as observed in a case of common rheumatic pericarditis. The process usually commences in the region of the great heart vessels, but very soon spreads to the whole surface: it is seldom limited to the starting point. This would seem to indicate that the irritant is carried hither and thither in the sac by the motions of the heart and natural currents of the serous contents (Coats).

As seen with the **naked eye**, the first thing to happen is an injection of the membrane, attended with a disappearance of its natural gloss and a roughening of its surface. (As might be expected these phenomena can be but rarely observed, since the patient seldom succumbs at this stage). Then exudation from the engorged vessels follows, and serous fluid begins to accumulate in the sac. Accompanying this, there is a deposit of fibrin upon the epithelial surface—commonly that of the visceral layer. To begin with, this deposit is an exceedingly delicate layer, white and soft, and very easily separable; but as the inflammation becomes more intense, more fibrin is deposited, layer upon layer, until a thick mass or "false membrane" is formed. It is now irregular and shaggy in appearance, though still remaining white and very soft, and it cannot be entirely separated from the membrane. After a period of 48 hours from the commencement of the inflammation, the false membrane is thickened and adherent, and cannot be separated: so that the condition of the fibrinous exudation affords some clue to the duration of the process (Greenfield). But meanwhile, the sac has been gradually distended with the persistent serous effusion, which may come to obliterate entirely the potential cavity of the sac by interposing a continuous layer of fluid between its visceral and parietal layers. More usually, however, the epithelial surfaces come together at one or more spots, and this circumstance determines what is popularly spoken of as the "honeycombed" or "pine-apple" appearance of the fibrinous mass. The incessant motion of the heart appears to alternately make and break contact between the visceral and parietal layers at the spots in question, just as one might do with two pieces of bread thickly spread with very soft butter. It will be evident that this peculiar appearance will be most marked on those parts of the pericardium where the heart meets with most resistance to its expansion, hence on the anterior aspect where it touches the parietes. After some time, we will find the pericardial sac more or less obliterated by complete or partial adhesion of its two layers.

Microscopically.—In the early stage there is congestion of blood vessels in the deep layers of the pericardium and epicardium, with commencing leucocyte exudation around. The epithelium shows cloudy swelling, and the connective fibres throughout the membrane are also swollen. Some hours later, coagulable lymph is seen on the surface: it is almost translucent, and presents numerous small spaces which now contain a few leucocytes but very rapidly become filled with these cells, as well as with detached epithelial cells and numbers of red blood corpuscles. The leucocyte exudation has increased. After a few days, when the false membrane has become adherent, we notice that the connective tissue element has proliferated, especially near the vessels, and that granulations are in process of formation, *i.e.*, new vessels, accompanied by connective tissue cells, are shooting into the superficial layers of the pericardium and epicardium, and beginning to penetrate the false membrane. Leucocytes in great numbers are also seen. Later still, we observe the fibrin being partly absorbed and partly eaten up by the granulations, so that by and by a layer of granulation tissue occupies the place of the former fibrinous layer. The serous effusion is is now absorbed, and the visceral and parietal pericardium come in contact, resulting in more or less complete coalescence of the granulating surfaces. There then ensues a gradual conversion of the granulation tissue into connective tissue, so that the pericardial sac is wholly or partially obliterated.

The above is a description applicable to a typical case of pericarditis, but there is very great variation in the phenomena, depending on the exciting cause and other conditions. Thus, in the first place, the inflammation may lead to a simple serous exudation with little else, and so it is common to term this a **serous** pericarditis. Or, the leading feature may be a fibrinous exudation with little or no serous distention of the sac, and this is termed a **dry** or **plastic** pericarditis. Again, there may be extravasation of blood into the sac mixed with the serum, constituting **hæmorrhagic** pericarditis. This is rare, and occurs mainly after Bright's disease and in alcoholic subjects, also in cases of cancerous origin. Sometimes, the inflammation is of a septic nature, *e.g.*, following empyema, and then the pericardial sac may contain pus; such a condition is known as **purulent** pericarditis. It is also caused by the rupture of pyæmic abscesses of the heart into the pericardial sac. Then, where the condition is associated with tuberculosis, the combination is named **tubercular** pericarditis. This may either be primary, or secondary to tubercle in the lung and pleura. In either case, there is very little serous effusion, but this usually turns purulent and then caseous. In the primary form, tubercle follicles are found amongst the granulation tissue. In the secondary form, which is more chronic, we find groups of tubercle follicles arranged round a caseous centre, and giant cells can be distinguished in them.

Results of Pericarditis.—If the irritation subsides within a day or two, the serous exudation and coagulable lymph may be entirely absorbed, and the membrane regain its normal appearance and structure. But such an issue is very rare. The usual course is for adhesions to form between the visceral and parietal layers, as already explained.

This is attended in chronic cases with more or less thickening: occasionally there is thickening without adhesion. Such thickenings constitute the "milk spots" which we recently considered. Then, as we have already seen, a serous pericarditis occasionally becomes purulent; the pus may afterwards caseate and ultimately calcify. Again, pericarditis may extend in the direction of the heart, or, on the other hand, outside the sac. In the first case, a superficial myocarditis results, which may extend deeply and cause serious damage to the heart wall, as we shall afterwards see. In the second case, cellulitis is set up in the mediastinum, and adhesions form with surrounding parts. Further, a common result of pericarditis is hypertrophy of the heart, and this can be explained in several ways; but it will be more convenient to defer the explanation until the subject of cardiac hypertrophy is taken up.

TUMOURS

Of the pericardium are nearly always **secondary.** The most important ones are **melanotic sarcoma, cancer,** and **fibroma.** Sarcomas most frequently spread from the mediastinum.

B. The ENDOCARDIUM.

HÆMORRHAGES.

In cases of septic poisoning, minute extravasations of blood have been found in the endocardium, and usually in its deeper part They are most common in the left ventricle. Probably they result from plugging of the vessels with bacteria.

FATTY DEGENERATION

Of the endocardium is nearly always associated with a similar condition in the heart wall.

Causes.—(1) Wasting diseases, *e.g.*, leucocythæmia, pernicious anæmia, and Addison's disease. (2) Poisons, especially phosphorus, arsenic, antimony, and alcohol.

Characters.—The degeneration tends particularly to attack the left ventricle, and is oftenest seen on the papillary muscles and the anterior cusp of the mitral valve. It produces patches which are paler than the healthy membrane, and may be tinged of a yellow hue. A very common **result** of the condition is calcification.

WAXY DEGENERATION

Affects the endocardium, in common with many other parts, in cases of general waxy disease.

Characters.—The situation of the change is not constant, but it involves the auricles more frequently than the ventricles. The elements affected are chiefly the capillaries, which are sparsely distributed in the deeper layers of the membrane; in advanced stages, the connective tissue fibres also become waxy. To the naked eye, the appearance is not characteristic; and we have to rely upon staining with iodine to diagnose the condition.

URATIC INFILTRATION.

Deposits of urates of sodium have been observed on the mitral and less frequently on the aortic valves, involving their endocardial covering. The deposits occur in cases of gout. They resemble calcified material, and might be mistaken for evidence of calcareous degeneration, but the application of the Murexide test at once distinguishes between the two conditions.

ENDOCARDITIS,

Or inflammation of the endocardium, may assume one of three forms—

I. Simple Acute Endocarditis; also called "warty" or "vegetative" endocarditis. This is the common form.

II. Ulcerative Endocarditis; primary or secondary.

III. Chronic Fibrous Endocarditis; usually secondary to I. or II., sometimes primary.

Causes.—These are very much the same as the causes that give rise to pericarditis. Acute rheumatism accounts for the vast majority of acute cases; Bright's disease for most of the remainder. Acute pneumonia, pyæmia, and septicæmia furnish a small percentage of cases.

Sites.—Endocarditis in the adult almost invariably manifests its effects on the left side of the heart, very seldom on the right side. In fœtal endocarditis, on the other hand, the right heart alone is usually attacked. Further, both in the adult and fœtus, the results are most frequently evident in connection with the heart valves. The reason of these limitations appears to be that the exciting irritant acts specially on such parts as are exposed to greatest friction—either from contact with the blood or with one another. We know that in acute rheumatism the special irritant "affects the joints where the synovial membranes lie against each other, and in the movements of the joints are moved on one another. It is as if, in addition to the irritant in the blood, the mechanical irritation of friction were necessary to the occurrence of inflammation" (Coats). It can now be readily understood that in adult life the mitral valve is most commonly affected, and on its *auricular* surface; less frequently the *ventricular* side of the aortic valve. Probably the venous state of the blood, which constantly obtains in the right heart during adult life, exerts a controlling influence on all inflammatory processes there.

I. SIMPLE VEGETATIVE ENDOCARDITIS.

Characters.—Along the line of contact of the valvular cusps swelling and thickening first occur, accompanied by thickening of the chordæ tendineæ. The parts appear translucent and gelatinous, though that may be obscured by blood staining, which renders them pink. Later, the cusps become roughened, and successive layers of fibrin are deposited on them, until a fringe of small papules or warts occupies the lines of contact. These warts are soft, friable, semi-translucent, and non-vascular, though sometimes blood-stained. The chordæ tendineæ are now greatly swollen and very brittle. The papules continue to increase in size, coalesce to form masses which are opaque and granular-looking, and when detached expose a rough, ulcerated surface. **Microscopically,** a fully developed vegetation closely resembles granulation tissue, but it contains no blood-vessels. The base of the growth is largely composed of a mass of small round cells, which are derived from proliferation of connective tissue cells in the endocardium. They stain deeply with picro-carmine. On the top of these there is some granular debris, which appears yellow with the same stain; while, at the margin of the growth, there is a thin layer of fibrin that takes on a pink stain. A few leucocytes may be found scattered through the granulation. If we stain a vegetation with methyl-aniline violet and examine under a high power, numbers of micrococci can often be seen. Probably they are of subordinate importance, and only accessory; they are not of the virulent kind found in the ulcerative form of the disease.

Results.—These depend to some extent upon the valve affected. There is very rarely a return to normal. Commonly, the process is prolonged until the new growths are converted into fibrous tissue; in the case of the mitral valve, this leads to adhesion of the cusps and narrowing of the auriculo-ventricular orifice (mitral "stenosis"). The chordæ tendineæ participate in the fibrous metamorphosis, and consequently often become shortened. This of course will prevent the valve from closing perfectly, and so bring about the condition of "incompetence." On the other hand, the valves instead of becoming adherent may shrink, so that the cusps cannot meet; this is a frequent outcome of aortic endocarditis, and causes incompetence of the aortic valve.

But very different sequelæ may also be got. The valves frequently become softened by the inflammation, and in their consequent weak condition they give way and stretch under the force of the blood current. This may be followed by (*a*) rupture, (*b*) ulceration, or (*c*) aneurism. When a cusp ruptures, the valve necessarily becomes incompetent. In the case of ulceration ensuing, pieces of vegetations are often broken off and are carried to other organs, producing embolism. These organs are chiefly the spleen, kidney, and brain; and they become the seat of simple embolic infarction. (We shall immediately see that emboli from *ulcerative* endocarditis invariably produces embolic abscesses). Sometimes the softened valve continues to yield beneath the blood pressure, gradually thins out and bulges so as to form a small bag, depending from the valve, and this constitutes aneurism of a heart valve. The commonest seat of such an aneurism is the aórtic valve, and the sac bulges into the ventricle—as one might naturally expect, from the direction in which the blood presses upon the valve. If it were on the mitral valve, it would protrude into the auricle. The aneurism may in course of time rupture, and in consequence of the perforation a fragment of the valve may become loose and hang down into the cavity. This by successive deposits of fibrin will sometimes form a long fringe, which eventually touches some part of the endocardium, and this becomes the starting point for a new process.

II. ULCERATIVE ENDOCARDITIS.

As we have seen, this may be a sequel of vegetative endocarditis and its leading **characters** have been mentioned. But the disease also occurs in a *primary* form, and possesses special features which justify its classification apart from the secondary variety. These features are mainly five in number. (1) Primary ulcerative endocarditis is due to septic and specific influences, for it occurs in pyæmia, acute pneumonia, malarial fever, puerperal fever, &c. (2) Its local action is more rapid, destructive, and widespread than in the simple form. The heart wall sometimes becomes involved, and has even been perforated. The inter-ventricular septum especially has repeatedly been ulcerated through at the upper part. Or, an abscess may form in the heart wall, and lead to an acute aneurism of the wall. (3) Nor does it confine itself to the left side of the heart: the right heart is frequently affected. (4) The fragments of vegetations which are carried into the circulation give rise to embolic abscesses in the organs which harbour them. (5) In the ulcerated vegetations colonies of micrococci are found. Similar organisms can be observed in great numbers in the embolic abscesses. **Microscopically,** the vegetations have a general resemblance to those of the simple disease, but there is added the ulcerated surface, with crowds of micrococci in the coagulated fibrin which forms the margin of the growth.

As in simple endocarditis, so in the ulcerative form, aneurism or perforation of a valve may happen. But the disease never heals, however produced; and the true primary form is always fatal.

III. CHRONIC FIBROUS ENDOCARDITIS

Commonly follows on the acute form and, like it, is associated with acute rheumatism. The primary process simply continues, with modified intensity, and there is a gradual conversion of the granulations into connective tissue, which slowly extends to the remaining structures

Primary Ulcerative Endocarditis – always fatal

1. due to some specific influences
2. Local action more rapid, destructive & widespread than
in simple form
3. often also affects right heart
4. Detached fragments cause embolic abscess
5. Micrococci are present in large numbers.

of the valve. The immediate result is a great thickening of the valve and its attachments, with coincident shrinking. This may lead to incompetence. On the other hand, adhesions occasionally form, and this causes stenosis. But very often secondary changes occur in the valves: they become fatty, and then impregnated with lime salts. "This condition is of such frequent occurrence that it may be regarded as the normal termination of ~~myo~~carditis." The valve is now rigid, and if a large piece of it be broken off—as sometimes happens—the resulting embolism may be of serious consequence.

ena

There is a form of chronic endocarditis which is closely allied with atheroma. It most frequently affects the aortic valve, when we find simultaneous atheroma of that vessel. Like atheroma, therefore, this disease occurs in advanced life, and it attacks those whose occupations entail much strain on the circulation. The morbid alterations produced are very much like those mentioned in relation to the secondary variety, but perhaps they are less marked than the latter.

LESIONS OF CARDIAC VALVES AND THEIR EFFECTS.

(A.) SINGLE LESIONS.

I. Mitral Valve.

(1) **Mitral incompetence**, usually caused by puckering of the valves, and shortening and thickening of the chordæ tendineæ. The blood is permitted to regurgitate from the ventricle into the auricle at each systole. Consequently (*a*) the left ventricle hypertrophies to compensate for what is taken off its propelling effect by the regurgitation. But, more important (*b*), the left auricle dilates and hypertrophies. This gradually tells upon the pulmonary circulation, so that (*c*) the right ventricle, and subsequently the right auricle, dilates and hypertrophies. The most prominent phenomena are the dilatation of the left auricle and hypertrophy of the right ventricle, causing the heart to assume a somewhat quadrilateral shape.

(2) **Mitral stenosis** is commoner than the first lesion. It occurs very often in the form of a funnel-shaped tube, caused by fusion of the edges of the valves. The funnel points into the ventricle, and the apical opening is much smaller than the normal orifice. Another form of stenosis is that known as "button-hole mitral": here the cusps are thickened and tense, and fused to a greater or less extent at the ends of their free margins, so forming a small slit. The stenosed orifice interferes with the free passage of blood into the ventricle. The *effects* of this are that (*a*), in the first place, the left auricle dilates and also hypertrophies. This will lead to engorgement of the pulmonary vessels, and, as a necessary consequence (*b*), the right ventricle dilates and, requiring to exert increased force, hypertrophies. The right auricle usually becomes similarly affected. Further, since the left ventricle is

receiving a meagre supply of blood, it not uncommonly happens that (*c*) the left ventricle atrophies from impaired function. This, however, is not always the case, because the increased force of the right ventricle may maintain the equilibrium to a large extent; and, besides, sometimes there is an associated condition of aortic incompetence, which throws increased strain on the left ventricle.

II. Aortic Valve.

(1) **Aortic incompetence.**—This is a more common condition than aortic stenosis, and it is usually due to a thickening and shrinking of the cusps, which thus cannot meet perfectly to close the orifice. When the cusps are stretched to their utmost, they leave a triangular sort of opening in the centre. At the end of each systole there is a reflux of blood from the aorta into the ventricle. In consequence of this (*a*) the left ventricle is stimulated to increased effort to expel the surplus of fluid, and so it hypertrophies, and the hypertrophy is always accompanied by dilatation. This may of itself compensate for the aortic lesion. But the enlargement of the ventricle often entails a stretching of the mitral orifice, leading to incompetence, so that in course of time (*b*) the left auricle may be implicated. The change in the ventricle imparts a more conical shape to the heart. The inter-ventricular septum is often pushed well into the cavity of the right ventricle, so that on transverse section that cavity is crescent-shaped.

(2) **Aortic stenosis,** pure and simple, is an uncommon condition, being usually associated with incompetence. It prevents the complete emptying of the ventricle during systole, so that that chamber comes to be overfilled. As a result, the ventricle is reflexly stimulated as before to increased contraction, and undergoes enormous hypertrophy. This will generally produce satisfactory compensation; but when the heart muscle is unhealthy it may be unable to hypertrophy quick enough, and so becomes more or less distended. Following upon this, the left auricle becomes embarrassed, and latterly "we may have all the evil consequences of mitral disease."

It is to be noted that while mitral lesions affect the general circulation mainly by producing venous engorgement, aortic lesions primarily involve the arterial system.

III. Tricuspid Valve.

(1) **Tricuspid incompetence** is the commonest valvular lesion in the right heart. It may result from fœtal endocarditis, but more frequently it is brought about by the great dilation of the right ventricle following upon mitral disease or (as we shall presently see) pulmonary obstruction. The condition leads to dilatation of the right auricle, and a pulse wave is projected into the veins of the neck.

(2) **Tricuspid stenosis** rarely occurs alone; it is almost invariably

combined with mitral stenosis. So we shall refer to it when considering double lesions.

IV. Pulmonary Valve.

Pulmonary stenosis is the only important lesion of that valve, and even it is uncommon. It is usually a fœtal condition, and it has the effect of deranging the normal developmental changes in the heart and large vessels (*vide* "Malformations of Heart"). The most common cause of pulmonary stenosis is fœtal endocarditis, causing adhesions of the valve segments. We shall presently consider other forms of pulmonary stenosis not springing from valvular lesions. In the adult, pulmonary stenosis gives rise to hypertrophy and dilatation of the right ventricle.

(B.) DOUBLE LESIONS.

(1) **Aortic incompetence** is often associated with **mitral incompetence.** Both may simultaneously result from rheumatic endocarditis, or mitral incompetence may follow the other when the left ventricle is much distended as a result of aortic incompetence. In the latter case, the consequences of mitral incompetence are often delayed and usually less pronounced than in the former. In any case, the left ventricle is subjected to great strain.

(2) **Mitral stenosis** and **tricuspid stenosis** nearly always co-exist, as already noted. The reason of the combination is unknown. Curiously enough, the heart frequently suffers no detriment from the lesions, the effects of one lesion seeming to balance those of the other. After a time, there may be hypertrophy and dilatation of one or other cavity.

INTRA CARDIAC THROMBOSIS.

Thrombi in the cavities of the heart are usually a sequel of endocarditis, and their formation depends upon two factors mainly, (1) damage to the lining membrane, (2) stagnation of blood in the chambers. We have just seen how endocarditis sets up changes in the valves, and very often leads to dilatation of the chambers. Sometimes this dilatation is compensated by the process of hypertrophy, but frequently the heart wall is left weakened, so that its function can only be carried on in a feeble manner. The consequence is that the contained blood is imperfectly emptied from the various cavities at the end of each systole, and so more or less stagnation occurs. The latter will naturally take place principally at the apices of the ventricles and in the apendices of the auricles when it involves these respective chambers. Now, since dilatation of the right heart is more common than of the left, it might be expected that thrombosis would occur more frequently in the right side than the left; and such is the case, although thrombosis in the left side is common enough. Further, thrombosis usually begins in the auricles.

On the right side of the heart, the commonest position for a thrombus is the *right auricular appendix*, and it usually takes the form of a "globular thrombus." Occasionally, however, it assumes a polypoid form, and protrudes into the sinus venosus, where it may hang free. Portions of such a thrombus are liable to become detached and form pulmonary emboli. On the left side, thrombosis of the *left auricle* is a pretty frequent result of mitral stenosis. As a rule, a thrombus in this situation grows to a large size, and in some cases we find the chamber quite filled, with the exception of a small central channel through which the blood merely trickles. Occasionally, aneurism of the auricle may be traced to thrombosis. Next in order of frequency the *apex of the left ventricle* becomes the seat of thrombosis. This is oftenest observed when the ventricle is greatly dilated. The thrombus is at first confined to the depressions between the columnæ carneæ, but in course of time grows into the polypoid form. The name "cardiac polypi" is sometimes applied to these growths, but it is not a correct term to employ. To return to the right side of the heart, the *right ventricle* is a common seat of thrombosis, and the thrombus sometimes extends into the pulmonary artery.

Characters of Thrombi.—It is necessary to distinguish between thrombi and *post mortem* clots, and generally this is an easy matter. Clots formed after death are dark red in colour, soft, gelatinous, transparent, and moulded to the shape of the containing cavity, to whose walls they do not adhere. Those produced *in articulo mortis* may be more or less yellow in colour, because the slower formation has allowed many of the red corpuscles to sink and leave the higher parts of the clot pale, just as we get a "buffy coat" on the surface of blood coagulated outside the body. Otherwise, the two kinds of clot are quite similar. Thrombi, on the other hand, being produced during life, form more or less slowly, with the result that they have a white or pale yellow colour. They are moreover opaque, firm, dry, and elastic, and adhere tenaciously to the heart wall; and when torn across, a corrugated surface is exposed. Under the microscope, they show a larger proportion of white cells than the *post mortem* clots. But sometimes thrombi have a red colour—when recent—and are then to be distinguished from red clots by their other characters. Again, thrombi may be overlaid and concealed by *post mortem* coagulation, thus rendering them liable to be mistaken for simple clots.

C. The MYOCARDIUM.

Under this heading we shall consider the *structural alterations of the heart muscle.*

HYPERTROPHY.

In studying *valvular lesions*, we saw that a constant sequel of these

lesions is hypertrophy of the heart, usually attended with dilatation. We saw also that, though in particular cases individual chambers may undergo hypertrophy, yet the condition very often becomes general in course of time.

Other causes of cardiac hypertrophy have now to be noted. In the first place, *pericarditis* very commonly interferes with the action of the heart to such an extent as to induce hypertrophy of the organ. This interference may come about in more ways than one. Thus, the mere inflammatory exudation in the earlier stages may so embarrass the heart as to lead to increased effort and consequent hypertrophy. Again, the superficial myocarditis which is sometimes set up weakens that part of the wall affected, and the deeper healthy portion is prompted to increased exertion, leading to hypertrophy. But further, and more important than either, an adherent pericardium—especially when the adhesion is outside as well as within the sac—offers great resistance to the full contraction of the heart, and necessitates extraordinary force on the part of that organ to expel its contents at each systole. The consequence is that very great hypertrophy may ensue. As in valvular disease, pericarditis causes a *general* hypertrophy of the heart.

Secondly, in *chronic Bright's disease*, there occurs a form of cardiac hypertrophy, affecting purely the *left ventricle.* The heart in consequence becomes more elongated in shape, the apex especially being markedly pointed. On making a transverse section of the chamber *post mortem*, the wall is found to be greatly thickened, while the cavity is apparently smaller; this is very often termed "concentric hypertrophy." (Where thickening of the wall is combined with enlargement of the cavity, the term "excentric hypertrophy" is similarly applied.) Various theories have been put forward to account for this hypertrophy of the left ventricle in renal disease; and the most likely one is that it is directly due to increased pressure in the systemic arteries. Greenfield ascribes it to the "resistance of the peripheral blood-vessels, which is no doubt due to the accumulation of urea in the blood." A second theory is that the heart is excited to hypertrophy by direct contact with some effete substance, which is supposed to accumulate in the blood during the course of the disease.

In the third place, *disease of the lung*, leading to obstructed circulation, often causes hypertrophy of the *right ventricle* alone. The disease emphysema may be specially instanced; as will be afterwards pointed out, that disease is associated with obliteration of many of the pulmonary vessels, and since the same amount of blood must then pass through a smaller number of vessels, it is obvious that "vis a tergo" must be increased. This, of course, tells on the right ventricle, which accordingly hypertrophies. That hypertrophy is never extreme, and is nearly always accompanied with dilatation. The right ventricle does not have the same capacity for hypertrophy as the left: this fact is no doubt largely dependent on the relative thicknesses of their walls. The chamber becomes altered in shape, and the heart assumes a quadrilateral form, its apex being abnormally blunt.

Fourthly, a frequent cause of cardiac hypertrophy is *aneurism.* An aneurism of the aortic arch takes away from the efficiency of the heart in forcing blood through the systemic arteries, and consequently the left ventricle undergoes compensatory hypertrophy.

A certain degree of cardiac hypertrophy occurs with *advancing age.* Doubtless it is compensatory in nature, the organ having to put forth extra effort in order to compensate for the loss of contractility in the arteries.

ATROPHY

Of the heart takes place under conditions which give rise to general atrophy, or emaciation of the body. Thus, in cases of cancer, stricture of the œsophagus, and tuberculosis, the organ is found atrophied after death. But phthisis does not always cause atrophy of the heart, for the circulation through the lung being more or less obstructed in that disease, the right ventricle often hypertrophies, for a reason that has been already mentioned.

BROWN ATROPHY.

In emaciating diseases, such as those just mentioned, the heart sometimes atrophies, and at the same time the muscle fibres assume a dark brown colour. The same phenomenon has been frequently observed in a senile heart.

This condition of "brown atrophy" possesses certain special **characters.** The heart, in the first place, is usually smaller, and it presents a dull brown colour, perhaps mottled. The coronary arteries are tortuous and stand out prominently on account of the loss of subpericardial fat. The muscular tissue is more flabby than natural, and rather friable. These features, however, are not characteristic of the atrophy, but denote fatty changes. When there is no fatty degeneration in the muscle, it is found dry and tough. **Microscopically,** the individual muscle fibres appear wasted, and no doubt they are abnormally thin. They are easily broken up into their constituent cells, the striation of which also is very noticeable. Around the nucleus, in an early stage, we observe a cluster of golden-brown pigment granules, most marked towards the poles of the nucleus. This, of course, merely indicates an exaggerated degree of a normal process, for it will be remembered that in the healthy muscle cell a small amount of pigment is deposited in relation to each pole of the nucleus. But in an advanced condition, the pigment is much more widely distributed; it is deposited in elongated patches following irregularly the lines of longitudinal

striation, and occupying a portion only of the breadth of the cell. The nucleus is now quite obscured.

The source of the pigment is unknown. Probably it is a form of blood pigment, but whether fresh pigment is actually deposited from the blood has not been determined. Pigmentation of the heart, further, is not confined to atrophied organs, it may be present in any hypertrophied heart (Woodhead).

CLOUDY SWELLING.

The heart is an organ that constantly shows this change, under those conditions which have been enumerated as the causes of cloudy swelling elsewhere. It usually denotes commencing inflammation or fatty degeneration.

Characters.—To the **naked eye**, no very pronounced alteration may be apparent, perhaps only a little pallor at parts—shewing a dirty grey tinge. Usually, the organ lacks firmness, and may be even friable : the wall can then be broken down by pinching between the fingers. On section, pallor is predominant, combined with some translucency. Under the **microscope**, the muscle fibres are seen to be opaque and swollen. Each muscle cell is crowded with minute granules, which obscure the nucleus as well as the striæ to a large extent. Sometimes, the interstitial connective tissue shows evidence of proliferation : this is so when the process is an incipient inflammation.

FATTY DEGENERATION.

Causes.—Any condition involving a serious deterioration in the quality of the blood is prone to set up fatty degeneration in the heart muscle. A convenient classification of causes can be made if we bear in mind that this degeneration may vary in degree. Thus, a very pronounced and often rapid process results from (1) poisoning by phosphorus, arsenic, etc., or (2) wasting disease, such as pernicious anæmia, acute phthisis, and Addison's disease, or (3) septic and infectious fevers. On the other hand a minor degree of fatty degeneration may be the sequel of (1) chronic alcoholism ; (2) chronic phthisis and other slowly debilitating diseases ; (3) hypertrophy of the heart ; the immediate cause of death in cardiac hypertrophy is often this fatty degeneration (Coats) ; (4) diminished blood supply from partial occlusion of the coronary arteries by atheroma or other lesion.

Naked eye char.—These depend upon the acuteness of the process. *(a)* In a fatal case of phosphorus poisoning, the heart on examination proves to be flabby, somewhat friable, and paler than normal. Pallor is also apparent on section, especially towards the inner surface. Indeed, the change chiefly affects the inner layers of the muscular substance, and an inspection of the endocardial surface reveals those characters which are most striking. It is seen that the change is limited to the left ventricle, involving the columnæ carneæ and musculi papillares, as well as the wall generally; and that these parts are marked with yellow spots and streaks, or patches, contrasting strongly with the remainder of the muscle. In addition, small hæmorrhages may be observed beneath the endocardium. The base of the ventricle is more markedly affected than the rest, probably because it is at the base that the blood exercises greatest friction (Greenfield). *(b)* When the degeneration is *chronic*, the heart becomes dilated, and it is not so firm as normal. Externally its colour is but slightly altered, being a shade paler, but on the endocardial surface we note a typical appearance. There are numbers of small cream coloured areas dotted over the surface so as to resemble a "thrush's breast." In an advanced stage, these areas have spread so as to produce an appearance which has been compared to that of a "faded leaf." Like the acute degeneration this minor form tends to involve the inner layers of the muscle; but unlike the former, this affects the entire heart, not limiting itself to one chamber, although the left side suffers more than the right.

Micro. Char.—These vary somewhat with the rapidity of the change. Under a low power (× 50) the degenerated patches are opaque, contrasting with the transparent healthy muscle. A high power (× 300) shows that the sarcous matter of the muscle fibre has been replaced to a greater or less extent by minute droplets of oil. This transformation may be seen to commence in the vicinity of the nucleus and spread toward the periphery of the cell. The fibres shrink and lose their normal transverse striation, but when there is an inflammatory origin, the fibres may be swollen. In chronic cases the fat droplets arrange themselves in a linear fashion along the axis of the fibres; in acute cases the deposition of the oil is less uniform, being distributed irregularly through the cell, and only showing traces of linear arrangement here and there.

It will be observed that the microscopic characters bear a certain resemblance to those of cloudy swelling, and we can only satisfactorily distinguish between the two conditions by applying the ordinary chemical and staining reagents.

FATTY INFILTRATION.

The heart normally presents a certain amount of fat upon its surface, lying beneath the pericardium. This sub-pericardial fat is most abundant along the course of the coronary arteries, at the origin of the large vessels, along the lower border of the right ventricle, and at the apex of the organ; the surfaces of the ventricles are usually free.

Fatty infiltration of the heart manifests itself first in an increase of sub-pericardial fat, causing the organ to appear larger. As the condition progresses, this accumulation becomes greater, and the fat gradually

infiltrates the heart wall. The entire thickness of the wall is not commonly involved, merely its outer layers, and these become atrophied and flabby; the inner layers remain fairly normal. If we examine the heart muscle under the **microscope** we find streaks of adipose tissue extending from the surface into the connective tissue between the muscle fibres. The connective tissue corpuscles are filled with oil, and look like fat cells. The muscle fibres are compressed and atrophied; they may disappear entirely and be replaced by adipose tissue.

As a result of these changes, the muscular wall of the ventricles may become largely replaced by fat, the inner part of the wall alone remaining healthy. This of course means great impairment of function. In cases of general obesity, the heart often suffers damage in this way. But in other conditions there is reason to believe that the fatty infiltration is not the cause of the impaired function, but a *sequel* to it. We know that when a voluntary muscle loses its function, it becomes infiltrated with fat. In the same way, therefore, we may infer that in the existence of general debility or wasting disease—involving a loss of function in the organ—fatty infiltration supervenes as a secondary change.

OBSTRUCTION OF ARTERIES.

The coronary arteries are rarely obstructed by emboli, probably because their direction is unfavourable to foreign matters gaining access to them. Vegetations from ulcerative endocarditis occasionally get impacted in the coronaries. When they lodge in a terminal branch, infarction of the heart wall may result. Obstruction of the main arteries and consequent mal-nutrition of the organ sometimes gives rise to a condition called **myomalacia cordis**. This essentially consists in a degeneration and absorption of the muscle fibres, resembling very much Zenker's degeneration. After a time, there is a proliferation of connective tissue, and consequent cicatrization.

Localized fibroid patches are sometimes discovered in the heart wall, and these have been attributed to obstruction of the coronary arteries by atheroma, which may sometimes be of syphilitic origin.

MYOCARDITIS.

This means inflammation of the heart wall. The muscular tissue, however, is never alone affected; there is invariably some interstitial change, involving the connective tissue elements between the fibres. The degree of interstitial change varies with the nature of the inflammation.

Three **varieties** of myocarditis may be mentioned, corresponding closely with those of endocarditis, vizt. :—

I. Simple Acute myocarditis.
II. Suppurative myocarditis.
III. Chronic Interstitial myocarditis.

I. SIMPLE ACUTE MYOCARDITIS.

This rarely occurs as a primary condition, it is almost invariably secondary to endocarditis on the one hand, or pericarditis on the other. Therefore the causes of these two inflammations may be regarded as more remote causes of acute myocarditis.

Characters.—As might be reasonably anticipated, pericarditis does not lead to the same morbid phenomena as endocarditis. Thus myocarditis following inflammation of the pericardium generally involves the entire area of the heart wall, though only penetrating a short distance into its substance. On the other hand, endocarditis usually induces localized patches of inflammation in the heart wall, affecting its inner layers chiefly. Let us consider first then—

(1) MYOCARDITIS FOLLOWING PERICARDITIS.

In a typical case the superficial muscular tissue is attacked. If we could see the heart in a very early stage, the surface would be pale, and mottled with a red injection of the intra-mural capillaries. The fibre would also be swollen and soft. At a later stage the colour becomes of a deeper red, and the tissue is very soft and friable.

Microscopically, in the early stage, all that we can see is a swelling in the muscle fibres, which have lost their striation, an engorgement of capillaries, with perhaps some leucocyte exudation between the fasciculi. At an advanced stage, the muscle fibre has a glossy appearance, exactly like that of hyaline degeneration; or it may have become broken down, and exhibit a granular debris, which by suitable staining can be shown to be partly composed of oil droplets. Between the fibres there is a proliferation of cellular connective tissue, and leucocytes are observed in great abundance. The latter are mixed with the nuclei which have been derived from the proliferated connective tissue. The small vessels are dilated, and surrounded by leucocytes.

(2) MYOCARDITIS FOLLOWING ENDOCARDITIS.

Inflammation of the myocardium under these circumstances affects the *left* side of the heart. It involves the muscle on its internal aspect, and produces changes at one or more foci. A favourite seat is the musculi papillares, and it occurs less frequently at various localized parts of the wall. The changes, both naked eye and microscopic, are precisely similar to those in the first form.

Results.—If the inflammation is slight and soon subsides, the inflammatory products are absorbed, and the muscle may recover its healthy aspect. But very often the inflammation becomes chronic, thus merging into the third variety mentioned in our classification. Sometimes the acute inflammation becomes very intense, and causes softening of the heart wall: this is especially the case with that form following

endocarditis, and results in local softening of the wall. The softened patch naturally gives way before the blood pressure, until it bulges so as to form an "aneurism of the heart wall." We shall afterwards see that aneurisms of the heart wall may result from fibroid degeneration, in which connection they will be again referred to.

II. SUPPURATIVE MYOCARDITIS.

This is an acute septic inflammation, resulting from pyæmia or septicæmia. In about one-third of the cases of pyæmia there is suppuration in the heart (Coats). The condition is the result of emboli lodging in branches of the coronary artery.

Characters.– The inflammation is commonly localized, involving the base of the left ventricle on its posterior aspect—close to the auriculo-ventricular septum. We find in this situation intense congestion of the myocardium, with a series of small elongated abscesses in the midst of the congested area. They primarily appear as minute yellow points, shown in strong relief against the congested tissue; then they rapidly soften and suppurate. Under the **microscope,** we see the ordinary evidences of intense inflammation of the myocardium, with, in addition, pus and swarms of bacteria in the vessels. The organisms are seen to more advantage before suppuration has occurred.

Results.—These abscesses rarely have an opportunity of healing, because the patient quickly succumbs to the general disease. But possibly they might take on a healing process, if time permitted. When situated near the pericardium, they often rupture into the sac and set up purulent pericarditis. If situated beneath the endocardium, they may open into the cavity, and it is said that they may form the starting point of an aneurism.

III. CHRONIC INTERSTITIAL (FIBROID) MYOCARDITIS.

As we have seen, this process is very often a sequel to an acute myocarditis. It is frequently a result of syphilis, and also of increased tension or strain in a dilated or hypertrophied heart. Localized fibroid patches are sometimes got in the heart wall, when no obvious cause is apparent. This "primary" condition may be a simple inflammation, or it may result from atheroma of the coronary arteries, or possibly from infarction of the heart wall (Greenfield).

Characters.—(1) **Secondary** (*a*) When the condition *follows pericarditis* the outer layers of the heart muscle become replaced by connective tissue. Any remaining fibres are atrophied, or may be undergoing fatty degeneration. The inner thickness of the wall is intact, although it may show an increase of the interstitial tissue. The blood-vessels are thickened, especially in their adventitia. (*b*) In that form *secondary to endocarditis*, the most constant effects are observed in the papillary muscles. These are gradually replaced by young connective tissue, until they become quite fibrous, and consequently shortened. The action of the valves will be impaired on this account. (*c*) The fibroid change *associated with dilatation or hypertrophy* of the heart, is more or less general, because the tension or strain is evenly diffused.

(2) **Primary.** This localized fibroid degeneration is almost confined to the *left ventricle*, and it affects either the anterior or posterior wall in the lower two thirds of the chamber. It is very rare in the right ventricle or inter-ventricular septum. On examination, the wall of the left ventricle exhibits a number of sharply defined tough patches within the area defined. They are as a rule most marked towards the endocardial surface, and the endocardium frequently suffers damage so that a thrombus forms over the patch. But they sometimes extend to the pericardium and set up a chronic pericarditis, leading to adhesion over the patch. At other times, the fibroid patches are completely embedded in the muscle, so that they are only revealed on making a section. Occasionally, the patches are partly calcified, or the wall in their vicinity has given way so as to form an aneurism. Microscopically, the muscle fibres are totally or almost totally replaced by firm connective tissue, which is very tendinous and contains few corpuscular elements. The capillaries are thickened in their walls. The muscle fibres around the patch are often extremely fatty, particularly when there is aneurism of the wall.

The process of chronic myocarditis may be aptly compared to cirrhosis of the liver or kidney.

Results.—While a simple substitution of connective tissue for muscle is bound to weaken the heart in its function, the possibility of further changes renders the condition more serious. Of these the most important is a local dilatation or **aneurism of the heart wall.** We already saw that aneurism may be due to acute inflammation and softening (page 123), but it is a more frequent result of fibroid degeneration. Aneurisms are practically limited to the *left ventricle.* By far the commonest position for them is at the apex, oftener on the anterior than the posterior wall. Less frequently they occupy the inter-ventricular septum, and occasionally they occur in the upper part of the chamber. These aneurisms commonly grow so large as to cause considerable bulging externally : but in certain positions they produce no outward bulging, and then the wall is simply excavated. Occasionally, the blood burrows so extensively into the muscular substance as to produce what is known as a "dissecting aneurism." The ultimate fate of cardiac aneurisms is frequently rupture, blood escaping into the pericardial sac and quickly causing stoppage of the heart.

TUMOURS.

I. **Primary** tumours of the heart are exceedingly rare. **Fibroma** and **myoma** may possibly occur.

II. **Secondary** tumours are not so rare. The commonest is **melanotic sarcoma**, spreading from the mediastinum, and involving first the pericardium then the muscle. Sometimes a **cancer** of the œsophagus extends to the heart, and very often in conditions of diffuse cancerous growth, the heart is invaded by round tumours.

INJURIES AND RUPTURE.

Wounds of the heart are naturally serious contingencies, and are commonly fatal. But even a penetrating wound of the heart, involving the whole thickness of the wall, does not necessarily cause death ; if it does not divide the muscular fibre transversely, healing by connective tissue formation may occur. Further, stabs in the ventricles are more likely to heal than stabs in the auricles, in fact it may be said that penetrating wounds of the auricles are always fatal. The interlacing nature of the muscular fibre of the ventricles accounts for this occasional immunity, because they exert a sphincter like contraction round the orifice of the wound. Again, since the left ventricular wall is considerably thicker than the right, stabs through the right ventricle are oftener fatal than those through the left. Stabs penetrating the aortic wall are always rapidly fatal. Missiles such as rifle balls have been known to lodge in the heart wall and not kill the individual. Violent kicks over the præcordia have caused rupture of the valves, and in some cases of the organ.

Rupture of the heart has frequently resulted from aneurism and from fibroid degeneration, as previously mentioned. Occasionally, a rupture has occurred without any very obvious cause, further than some straining at the time, and is then termed spontaneous. Sometimes the wall has been discovered to be fatty in such cases, but this cannot explain all the instances. Very probably, the predisposing cause is a local softening of the wall consequent upon obstruction of some branch of the coronary artery (Greenfield).

II. The ARTERIAL SYSTEM.

ATROPHY.

When an artery is cut off from its area of distribution, and no longer conveys its normal quantum of blood to nourish the tissues, it gradually wastes by a process of simple atrophy. Thus after amputation of the leg above the knee, the lower part of the femoral artery atrophies. Similarly when a vessel is ligatured, there is more or less atrophy on the proximal side of the ligature, as far up as the nearest anastomotic branch.

In considering the pathology of *chlorosis*, it was mentioned that in a number of cases the lumen of the aorta is narrowed, and its wall wasted. This is probably a congenital condition.

HYPERTROPHY.

The vascular system is a wonderfully plastic one; new vessels are developed from pre-existing ones, and fully formed vessels increase in thickness and calibre with great readiness when the tissues demand an augmented blood supply. The process of granulation tissue formation affords a familiar example of the rapidity with which capillary branches can be produced by simple budding from the old vessels. Then when a large vessel is occluded, artificially or otherwise, we know that a collateral circulation is established by a union of the branches coming off above and below the seat of occlusion. The united branches enlarge under the stimulus of the blood current, until they are sufficiently patent to afford an adequate blood supply to the previously impoverished tissues. In this way capillaries become arteries, and very small arteries assume large proportions.

In chronic Bright's disease (particularly when the kidney assumes the "cirrhotic" type) there are certain general vascular phenomena which apparently result from the renal lesion. In the first place there is a rise in the systemic blood pressure, and this is followed by hypertrophy of the heart. But what immediately concerns us in this connection is, that the arterioles throughout the body undergo thickening of their walls. How this comes about is not very apparent. The general opinion is that it results from the increased blood pressure, although it is still more difficult to explain how the increased tension itself comes about. A less likely theory has lately received some support. It is this,—that there is a general lesion affecting simultaneously the arterioles and capillaries throughout the body, and leading to what Gull and Sutton call "arterio-capillary fibrosis:" that the renal change does not necessarily occur first, but on the other hand only participates in the general process; and that the increased blood tension is a secondary result of the process.

FATTY DEGENERATION.

The whole arterial system is liable to be affected with fatty degeneration. The condition produces phenomena differing with the site. A. In the **aorta** and **large arterial trunks**, the inner coat is the one mainly involved. If we examine the ascending part of the aortic arch in an old person, we very often see small yellowish streaks or patches, not raised above the surface. With the aid of the microscope, this appearance is found to be caused by a fatty degeneration of the connective tissue cells between the laminæ and also of the endothelium. The cells exhibit the ordinary appearances of fatty change. Occasionally, the condition is followed by a deposition of lime salts, forming calcareous plates which can be stripped of. (But calcification in the aorta is usually due to atheroma). B. In **medium sized arteries**, fatty degenera-

tion affects chiefly the middle coat, and destroys the muscular wall. It also occurs in old persons, and very commonly ends in calcification. C. In the **minute arterioles** and **capillaries**, the entire wall suffers. The capillaries of the liver and other organs are especially affected in cases of phosphorus poisoning and pernicious anæmia. The endothelial plates commence to degenerate around the central nucleus, and soon become crowded with fat droplets. The capillaries, as a result of the change, are weakened and readily rupture, giving rise to small hæmorrhages.

CALCIFICATION.

This is a very common sequel to fatty degeneration in the arteries in old age, and is nearly always seen in the middle coat. Lime salts are deposited in the muscular coat, often in the form of calcareous rings surrounding the vessel in the position of the middle coat. The wall in consequence becomes rigid and diminished in calibre. Thrombi are apt to form on the altered wall; and, more important, since the damaged vessels transmit less blood than normal, senile gangrene may ensue. Calcification often takes place on atheromatous patches.

Calcification has been observed in the arterioles of the brain and kidney, where there was no obvious preceding phenomenon. Further, a "metastatic" calcification of the inner coat of arteries has been described, partaking of the nature of infiltration rather than degeneration. The supposed origin of the lime salts in one case was an acute caries of the vertebræ.

HYALINE AND WAXY DEGENERATIONS

Are to a large extent diseases of arteries, so they will be found fully described in the "General" section of the book. It should be noted that waxy degeneration has not been observed in the aorta or its main branches.

ENDARTERITIS.

There is a close analogy between the intima of blood-vessels and the lining membrane of the heart, and, like the endocardium, the former is liable to the results of inflammation. This is accounted for by the constant exposure to irritation which the inner wall of an artery suffers by reason of its contact with the blood. The term "endarteritis" expresses an inflammation of the inner coat.

Classification.—There are two great types of endarteritis, viz.:—

I. ENDARTERITIS DEFORMANS.
  (1) *Acute.*
  (2) *Chronic.*
II. ENDARTERITIS OBLITERANS.

I. ENDARTERITIS DEFORMANS.

Causes.—These are mainly two in number—(1) alcoholism and (2) syphilis. Further, however, the disease is essentially one of advanced life, and is aided in its occurrence by any increased strain exerted upon the vessels from within.

(1) ACUTE ENDARTERITIS DEFORMANS.

This is a rare form of inflammation, and is only met with in the aorta, where it attacks the inner coat just beyond the valves.

Characters.—On the inner wall of the aorta, near the valves, we find a number of patches as large as a split pea, soft, slightly raised above the surface, and of a yellow or yellowish-grey colour. They are, it should be noted, very readily stained with blood, and so, *post mortem*, may appear pink. The surrounding surface is smooth and swollen, perhaps slightly wrinkled. Suppuration rarely occurs; but, when the condition is associated with endocarditis of the aortic valve (as it sometimes is), the inner coat of the aorta may become thickened or ulcerated. Occasionally, the soft patches yield before the force of the blood current and form "acute multiple aneurisms." There is evidence of concurrent periarteritis in the shape of swelling and softening of the outer wall. The **microscopic** appearances are just the same as those observed in an early stage of chronic inflammation, so, for convenience, they will be described in that connection.

(2) CHRONIC ENDARTERITIS DEFORMANS—"ATHEROMA."

This is an extremely common condition, and may affect the whole arterial system, capillaries alone excepted. It is prone, however, to attack the larger vessels, and it does so at the seat of greatest stress. For instance, in the aorta—the principal seat—atheroma most frequently occurs just beyond the valves, then around the origin of the great arterial trunks, next at the origin of some of the lesser branches.

Characters.—The process begins in the formation of patches similar to those produced in the acute variety; less commonly the change commences in more diffuse form. In the first case, the patches grow larger until fusion takes place here and there, resulting in the production of considerable areas of thickening. These are firm in consistence and smooth on the surface, showing that the endothelium is still present. Soon the patches show a yellow centre on section: later they become almost wholly yellow. Then they gradually become hard and gritty, evidently from the presence of calcareous matter. In a medium sized artery these patches are usually confined to one side of the artery, and they project in a nodular form into the lumen so as to diminish its calibre: a transverse section of the vessel therefore has the configuration of a signet-ring. Thrombi are often found adhering to the patches.

Microscopically, the changes are shown to be not purely inflammatory, but to partake largely of a degenerative type. First consider the incipient soft patches which are found in the very early stage. They consist practically of great numbers of rounded and flattened cells, which are derived from an active proliferation of the fibrous elements of the inner coat. The change does not extend beyond the internal elastic lamina. In those parts most thickened there is a great preponderance of the round cells. They stain deeply with picro-carmine. The endothelium is intact and covers the patches. The adventitia also shows increase in the number of its cells, so much so sometimes that it comes to resemble a mass of granulation tissue. Some of these cells originate by proliferation of pre-existing cells of the adventitia; others are derived from inflammatory exudation from the vasa vasorum.

Now examine a section of the aortic wall in a more advanced stage of the process. It usually happens that the inner coat alone is involved, and sharply defined from the middle coat. The fibrous laminæ are now swollen, greatly proliferated, as are also the elastic elements: numerous cells, flattened and granular, occupy the spaces between the swollen fibres, and stain but imperfectly with picro-carmine. These changes are most marked in the deeper layers of the intima. When the patches have become yellow, we find that the proliferated cells and, in turn, the fibrous matrix have undergone fatty degeneration. This is quickly followed by a deposit of lime salts in the connective tissue corpuscles especially, and then the appearance presented is usually that of a somewhat homogeneous matrix, in which are numerous spaces containing a granular debris with some crystals. The debris is composed of fat globules, calcareous granules, and cholesterine crystals. The calcareous granules by and by agglomerate, so that actual plates of the hard material lie deep in the inner coat and render the vessel rigid.

Sometimes endarteritis in the aorta does not assume quite such a chronic form, and then the internal elastic lamina may give way, and the middle coat and even the outer coat may be invaded by the degenerative process. The muscular fibres soften, become fatty, and may be partly absorbed or become calcareous like the inner coat. The softening may lead to dilatation.

In a medium sized artery the appearances are very similar, but there is less proliferation of the connective tissue elements, and the proliferated cells tend to be disposed in rows alternating with layers of the fibrous laminæ.

Sequelæ.—An atheromatous patch generally develops various further processes, leading to greater or less impairment of the vessel wall, and frequently to damage of the tissues through interference with the circulation.

(*a*) It has been stated that the smaller arteries have their lumen diminished in consequence of the nodular thickening of their walls. The same holds true in the aorta, but the relative diminution in that vessel is so slight as to be of little consequence. But in arteries like the coronary and cerebral arteries the interference with the blood flow may produce serious results. The interference is aided by the rigidity of the walls, which is a necessary result of calcification, when that has occurred: and it is often completed by the formation of a thrombus over the patch. This is a frequent cause of softening in sections of the brain supplied by the obstructed vessels.

(*b*) In the aorta rigidity and weakening of the wall lead to very important consequences. The vessel is unable to assist the left ventricle in propelling the blood through the body, and that primarily causes some stagnation in distant parts. The ventricle has more work thrown upon it, and consequently hypertrophies: the blood is thus projected into the aortic arch with abnormal force, dilating the vessel and producing a diffuse aneurism. Atheroma in the smaller arteries more commonly gives rise to thrombosis than dilatation.

(*c*) It is a very common thing for an atheromatous patch to soften and become transformed into a mass of debris confined by a slight capsule of connective tissue, constituting an "atheromatous abscess." The debris consists of fat droplets and cholesterine crystals with broken down cells. The abscess then ruptures, and becomes an "atheromatous ulcer." Particles of the debris may be carried away, and cause embolic plugging of small vessels. Further, the blood may find its way between the coats of the aorta, separating them up so as to form a "dissecting aneurism." The abscess does not always rupture however; its contents are sometimes absorbed, and this leads to a depression on the inner surface.

(*d*) The calcareous plates, which so commonly occupy the deeper part of the intima, not unfrequently tear through or erode the superficial layers, and present an edge or angle inside the vessel. A thrombus is very likely to form on this projection, and it may cause plugging in the case of a smaller vessel. In the aorta a thrombus frequently extends in a polypoid form along the vessel, and part of it is liable to become detached and form an embolus.

(*e*) Atheroma does not directly lead to hæmorrhage as a rule. But there is a modified form of atheroma which is supposed to be the special precedent of aneurism formation in small arteries, particularly the "miliary aneurisms" on the cerebral vessels; and these miliary aneurisms are now recognised as the chief cause of cerebral hæmorrhage.

III. ENDARTERITIS OBLITERANS.

Although this condition is classified along with atheroma, it is not an independent disease, but merely a phenomenon in other processes. It signifies a gradual occlusion of a blood-vessel by thickening of the

Sequelae of Atheroma

1. Interference c̄ blood supply to other parts
esp in small vessels.
2. Hypertrophy of left ventricle - Aortic Aneurism
3. Atheromatous abscess - ulcer - aneurism.
4. Thrombosis embolism
5. Cerebral haemorrhage from miliary aneurism

vessel wall. The thickening is limited to the inner coat, and in its nature resembles the less extensive thickening of atheroma before degeneration has set in. But it differs strikingly from the latter, inasmuch as the proliferated tissue becomes vascularized, so that the intima is converted into a dense mass of granulation tissue. Further, the inflammatory product does not caseate nor calcify.

We observe the process constantly in the healing of wounds. It is also well exemplified in the "organization" of a thrombus. Again, it is a very common occurrence in syphilitic lesions, affecting especially the cerebral arteries. It is usually met with in comparatively young people, thus contrasting with atheroma. On examining a vessel with syphilitic endarteritis, small nodular thickenings are observed at different points, some of them causing perhaps entire obliteration of the lumen. Under the microscope, the intima is seen to be the seat of the thickening, and this is mainly due to a great proliferation of the connective tissue cells amidst the laminæ and of the endothelial cells lining the vessel. There is usually some swelling of the outer coat, the result of cell infiltration, and this may go on to fibrous thickening. The contour of the vessel thus becomes irregular, and the term "periarteritis nodosa" is employed to express the particular change that has taken place in the adventitia. It is frequently associated with softening of the middle coat, leading to dilatation and aneurism. Lastly, obliterative endarteritis attacks the vessels in many interstitial inflammations., *e.g.*, in the lung and kidney. It will be referred to in these connections.

The injurious effects of obliterative endarteritis are most strongly manifested in the brain, where areas of cerebral tissue supplied by the occluded vessels soften and degenerate.

ANEURISM.

An aneurism proper means a localized and persistent dilatation of a blood-vessel. According to this definition, the wall or sac of the aneurism is a portion of the vessel wall which, however, may be altered both in thickness and structure. But it is necessary to bear in mind here that the term aneurism is commonly used by surgeons in a wider sense. According to them, it includes the result of hæmorrhage from a vessel when blood is poured out into the tissues, forming a cavity for itself, which becomes surrounded by a fibrous capsule. In such a case, while the encapsuled blood clot may lie close to a vessel, it does not necessarily communicate with the vessel and is in no degree surrounded by the vessel wall. These aneurisms are usually the result of a traumatic injury and so they are often spoken of as "*traumatic aneurisms*." The term "*spurious*" also is a general epithet applied to them, as distinguishing them from the orthodox or "*true*" aneurism which has been defined. The latter is further called "*spontaneous*," in allusion to its mode of origin.

Aneurisms may be classified according to their shape and extent. Thus, we speak of I. **Diffuse**, where a large part of a vessel irregularly dilates; II. **Fusiform** or spindle shaped, involving the whole circumference of the vessel; III. **Sacculated**, meaning a dilatation limited to one side of the vessel, and frequently having only a small orifice of communication with the vessel; IV. Cup shaped.

Cause.—There are two main factors in the causation of aneurism, vizt. :—(1) weakness of the vessel wall, and (2) increased blood tension. The wall of a vessel can be damaged in various ways, *e.g.*, by direct external violence, or by undue and constant pressure upon a vessel (*e.g.*, by a piece of fractured bone) leading to impaired nutrition of its walls. Again, emboli often cause damage of a vessel wall, especially in the brain. But by far the most important cause of damage is atheroma. In studying atheroma it was mentioned how frequently the condition terminated in the development of an aneurism; and it need only be added now that the degeneration and softening of the inner and especially the middle coat, following upon atheroma, is one of the chief elements which determine the dilatation. It must be pointed out, however, that while atheroma is a disease of advanced life, aneurisms usually occur before the age of 40. Further, aneurisms occur far more frequently in men than in women, and particularly in men who lead a laborious life, such as soldiers and mechanics. The preponderance of atheroma in the male is not so great. These facts point to the conclusion that the occurrence of aneurism is always associated with more or less strain, involving increased blood tension.

A further proof of this is afforded by a study of the **sites** which aneurisms are found to occupy. Speaking generally, aneurisms occur more frequently in the thoracic cavity than anywhere else. The thoracic aorta is the commonest seat of aneurism in the body. We know that that vessel is directly exposed to the effects of increased blood pressure when the heart's action is excessive. The first position affected in point of frequency is at the origin of the great vessels in the aortic arch: next to this is the ascending part of the arch close to the valves (Greenfield). Second to the thoracic aorta, the popliteal artery is the most frequent seat of aneurism. This artery has been known to develop an aneurism after an injury, caused, for instance, by a sudden bending of the leg in an elderly person. But, no doubt, the unique situation of the artery renders it liable to the effects of excessive blood pressure. Footmen, who are subjected to much standing (which exercises the gastrocnemii muscles), and who frequently wear very tight garters, are especially prone to contract popliteal aneurism. The compression of the artery causes increased tension above the seat of compression, and a prolonged strain of this nature may lead to weakening of the vessel wall, and ultimately to aneurism formation. After the popliteal artery, aneurisms occur most

commonly on the abdominal aorta and femoral arteries, then on the main branches of the aortic arch. Less commonly, aneurisms are found on the iliac arteries, on the arteries of the brain, and other internal organs.

Growth of Aneurisms.

In an atheromatous vessel, degeneration of the middle coat is the first indication of approaching aneurism formation. At the weakened part a slight bulging takes place, which gradually deepens into a pouch. Once started, an aneurism tends to enlarge indefinitely, for it is shown by a well known physical law that the blood pressure in the dilatation must be greater than the pressure at any other point of the vessel wall. The pouch therefore enlarges, and its mode of enlargement determines the future shape of the sac. Thus it may extend in all directions—circularly and longitudinally—until a fusiform or diffuse aneurism is formed ; or the pouch may become inflated, as it were, through the primary small opening, and this results in a sacculated aneurism. With regard to the structure of the sac, that never is the same as the arterial wall, unless the aneurism be situated on a small vessel. As a rule, the enlargement of the sac is accompanied by degenerative as well as formative processes in the sac.

Were we to trace the development of an aneurism, we should find that the muscular coat undergoes successive passive changes until it may ultimately disappear. Its fibres become stretched and dissociated, and at the same time degenerated, so that they cannot be seen except at the very margin of the orifice of the sac. The inner coat sometimes persists, although greatly thickened and altered by the atheromatous changes ; but very often it also disappears to a large extent, and can only be recognised at the margin, sometimes also here and there over the surface. In its place may be found a delicate layer of tissue having a hyaline appearance. The outer coat of the vessel is to be regarded as the only one constantly represented in the aneurismal wall. The adventitia is a connective tissue structure and, coincident with the dilatation of the vessel, undergoes inflammatory thickening and forms adhesions with surrounding parts. Thus the sac comes to be composed mainly of dense uniform fibrous tissue, which often reaches a great thickness.

If the circulation of blood through an aneurism be in any degree impeded, **thrombi** are deposited on the interior of the sac. It is evident that this phenomenon will occur with great readiness in a sacculated aneurism, where the orifice of the sac is relatively very small. The coagulated blood is deposited layer upon layer in the form of pale clots, constituting a "laminated pale thrombus." This is the state of matters in an early stage. The subsequent phenomena vary according to circumstances. The thrombus may continue to grow by successive accretions of laminæ so that the sac contains none but solid contents, and the circulation through it is completely arrested. Following upon this, organization of the thrombus sets in, and results in the conversion of the aneurism into a nodule of connective tissue. The aneurism may then be said to have undergone a natural cure, and it is the physician's aim in treating aneurism to encourage and hasten this natural process. However, such a favourable termination rarely occurs spontaneously, and then only in small vessels. The continual distension of the cavity appears to hinder the process of organization, so that the latter is likely to occur only when the cavity becomes entirely filled with clot, as above stated. Generally, the thrombus never entirely obliterates the sac. Very often, the blood finds its way between the thrombus and the wall of the sac, causing the former to be detached, so that it lies free in the fluid contents of the sac. Frequently, too, the blood insinuates itself between the laminæ of the thrombus, and after death we may find in such a case red thrombi alternating with white laminæ. When thrombi have existed for a length of time they become drier, firmer, and fibrous-like.

Terminations and Effects of Aneurisms.

Very rarely does an aneurism undergo spontaneous cure. We saw that it sometimes occurs through obliteration of the sac by thrombosis and subsequent organization of the thrombus. In other cases, it has occurred by the aneurism pressing upon the artery and occluding the lumen of the latter. This leads to obliteration of the sac as in the first instance. An identical result is obtained by the surgeon when he ligatures an artery on the proximal side of an aneurism.

The usual course of an aneurism is that it continues to enlarge until the sac gives way, when fatal hæmorrhage ensues. We shall presently see that a fatal issue is frequently the result, not of rupture, but of pressure upon surrounding parts. The rupture may come about in more ways than one. Thus, a rapid enlargement of the sac may cause such extensive thinning of the wall that it simply cannot resist the pressure, and gives way. Or the wall may become softened by inflammation and yield to the blood pressure, or the inflammation may induce sloughing first. The rupture is not always immediately fatal, for often the aperture is so minute as to admit of a leak only, and this may be temporarily restrained by clotting. These leakages perhaps recur many times in succession, and death may even result from the repeated draining of blood and consequent exhaustion. Commonly, however, the leaking aperture enlarges very soon and profuse hæmorrhage takes place, ending in rapid death.

Aneurisms produce injurious effects on surrounding parts by the pressure which they necessarily exert. In this respect they resemble simple tumours. These pressure effects will naturally be of a very serious nature when the aneurism is situated in such a cavity as the

emphysema

thorax, and in this relation it may be well to note the symptoms and lesions which **anenrisms on the aortic arch** may give rise to. In the first place, the heart suffers more or less. It is frequently displaced downwards by a large aneurism, the auricles being compressed to some extent. In addition, the organ hypertrophies to compensate for its impaired efficiency: the left ventricle chiefly is involved. Secondly, various vessels may be compressed, leading to vascular derangements. The superior vena cava has been completely obliterated in more than one instance, giving rise to excessive dropsical swelling of the face, neck, shoulders, and arms. The pulmonary artery is frequently compressed, leading to impaired blood supply to the lungs, and hypertrophy of the right ventricle. The pulmonary veins may be compressed, resulting in œdema and dropsy of the lungs, which is not uncommonly the cause of death. Again, the thoracic duct is sometimes subjected to pressure, causing engorgement of the absorbent vessels and glands, and resulting in inanition. Thirdly, nerves are often involved. Thus the recurrent laryngeal nerves are sometimes irritated, producing symptoms of laryngitis, with very marked stridulous breathing. Fourthly, the various canals within the thorax are all liable to suffer stenosis. Aneurisms on the transverse part of the arch, especially, press upon the trachea and impede the breathing; or on the œsophagus and cause the ordinary symptoms of stricture of that tube. Aneurisms involving the descending part of the arch frequently press injuriously upon the left bronchus, interfering with respiration. They often give rise to chronic broncho-pneumonia and ~~emphysema~~, a fact of great clinical importance. ~~[illegible] ?~~

When an aneurism involves an arterial trunk where the latter gives off branches, these are very often obstructed. The orifices of the branches may have been primarily narrowed by an atheromatous process, and this condition is aided by thrombosis in the sac. Or, the orifice may be so contorted by the increasing expansion of the sac as to prevent the free entrance of blood into the branch. Or the branch itself may be occluded by pressure of the aneurism upon it beyond the orifice. The result of closure of a branch is the formation of a thrombus in its interior, and its ultimate conversion into a fibrous cord. But instead of becoming obstructed, arterial branches arising at the seat of an aneurism may undergo dilatation, through the combined effects of the blood pressure and the traction exerted by the aneurismal wall.

Aneurisms have the property of promoting the absorption of parts with which they come in contact. Not only are the soft structures liable to be eaten through in this way, but even bone becomes eroded before the advancing wall. This process does not occur, however, without detriment to the sac itself, for eventually it gives way, and fatal hæmorrhage ensues. The hæmorrhage may find vent in various channels, depending on the site of the aneurism, and consequently on the direction of its pressure. Thus an aneurism on the ascending part of the aortic arch pressing on the pulmonary artery may in course of time establish a communication with the artery. Occasionally, the heart wall is opened into at some point. More commonly, an aneurism in that situation ruptures within its pericardial covering, and the sac of the pericardium is immediately distended with blood, the pressure of which quickly stops the heart's contractions, and causes rapid death. (Small aneurisms on the coronary arteries sometimes rupture and cause a fatal result in the same way.) Aneurisms on the transverse part of the arch very frequently open into the trachea and œsophagus, causing fatal hæmorrhage —in the one case into the air passages, in the other into the alimentary canal. Aneurisms involving the descending part of the arch not uncommonly rupture into the left bronchus and left pleural cavity. Or they may contract adhesions with the pulmonary tissue and destroy it to a certain extent, and so cause mortal hæmoptysis ; but such cases are not always fatal at once.

But, again, aneurisms very often extend until the sac comes in contact with the chest wall : this contact usually takes place either in front, with the sternum and cartilages of the ribs, or behind, with the vertebræ. Then commences a process of erosion of these hard parts, resulting in absorption of the portions involved. It is a curious fact that the comparatively soft cartilage resists more than the hard bone ; and in the case of an aneurism advancing to the front of the chest we may find the sternum and osseous ribs much eroded, while the cartilaginous ribs are very little destroyed. Similarly, when the vertebral column is attacked, the intervertebral cartilages remain unaffected.

Varieties of Aneurism.

Miliary Aneurism.—This name has been applied by Charcot to minute aneurisms situated on the smaller vessels of the brain. In nearly all cases of cerebral hæmorrhage occurring in persons over 50 years of age, such aneurisms can be found on the small vessels supplying the brain substance. They usually exist in considerable number in the same person, and their size may be stated as $\frac{1}{20}$ to $\frac{1}{12}$ of an inch. When examined microscopically, they are found to possess all the characters of ordinary aneurisms : they are usually fusiform in shape, sometimes sacculated. The cause of these aneurisms has not been determined, but the individuals in whom they are found are usually the subject of chronic renal disease, accompanied with hypertrophy of the left ventricle. They are not due to atheroma : possibly the explanation of their origin is a chronic periarteritis in the outer coat, resulting in sclerosis and leading to atrophy of the middle coat.

There are certain other lesions comprised under the name aneurism, although they do not come under the definition of the term, and these must be briefly noted.

(1) **Dissecting aneurism.**—This is a peculiar condition which is sometimes met with in the aorta, rarely in other vessels. It consists in a separation of the coats of the vessel by the blood, which gains access through some lesion on the inner wall. The commonest **cause** of the condition is atheroma, as before noted. An atheromatous patch just beyond the heart valves frequently ruptures, and allows the blood to plough its way into the wall. Generally, the fibres of the middle coat are separated, and the blood lies between the external and internal layers of the middle coat. The force of the blood stream frequently causes an extension of this unnatural channel for a considerable distance within the vessel wall, but usually terminating by a second rupture of the inner coat, which allows the blood to pass back into the vessel at a more or less remote point from the original rupture. Occasionally, the second rupture takes place through the outer coat, resulting in the formation of a false aneurism around. But as a rule the first method obtains, and so the blood comes to flow in two parallel channels, separated only by a thin septum. The new channel may in time become larger than the original one, and so the lumen of the vessel is narrowed.

Dissecting aneurisms are sometimes caused by injury, and occasionally they arise spontaneously. "There is no form of aneurism which is so frequently multiple as this."

(2) **Varicose aneurism.**—This means a condition when an aneurismal sac opens into a vein. Thus, an aneurism of the abdominal aorta has been known to establish a communication on its right side with the inferior vena cava. It will be readily understood that such a condition will tend to cause great venous engorgement.

(3) **Aneurismal varix.**—This term indicates a direct communication between an artery and vein. In the days when venesection was popular, it was a common enough thing, when opening a vein at the elbow, to wound the artery at the same time, and aneurismal varix was often the result.

(4) **Cirsoid aneurism.**—This, which may be termed an "arterial varix," means the condition in an artery resembling varicosity in a vein *(q.v.)*. It is often very well seen in the temporal artery.

(5) **Aneurism by anastomosis.**—This is somewhat allied to the former. It is observed mostly on the scalp, following a wound which involves a number of arteries. The vessels enlarge, lengthen, give off new branches, and form a pulsating mass resembling an angioma.

III. The VENOUS SYSTEM.

VARIX.

"Varix is dilatation of veins, just as aneurism is dilatation of arteries." The walls of veins are thin and weak compared to those of arteries, and readily stretch when increased strain is thrown upon them. Indeed, veins have an extraordinary capacity for enlargement to perform additional work thrown upon them. The normal blood pressure within veins is small, and increased pressure rapidly causes them to expand. So we must always look, for an explanation of varix, to some cause which is calculated to increase the blood pressure within the veins. Such causes are tumours, aneurisms (from without), and thrombosis (from within).

Morbid changes.—When obstruction first occurs, the vein dilates and exhibits isolated swellings, which indicate the site of the valves. The vein gradually increases in length, and so it is forced to throw itself into a sinuous form. This sinuosity goes on developing until the vein may show large swollen convolutions. The valves are naturally rendered incompetent by the dilatation, and this aggravates the condition. The progress of these changes can be easily followed in the leg of an individual when the superficial veins are involved. The blood stagnates in the varicose veins, and thrombosis not uncommonly occurs.

Microscopically, the affected veins are found to undergo a great thickening. The inner coat shows a laminated thickening closely resembling atheroma in an artery. The outer coat proliferates to some degree. The middle coat usually atrophies, and is often transformed into a calcareous ring so that the vein becomes rigid.

Sites.—(1) The *lower extremity* is an extremely common site for varix. Persons whose occupation necessitates prolonged standing are specially prone to the affection, doubtless because the muscular effort put forth in keeping the legs straight forces the blood into the superficial veins. A gravid uterus or tumour in the pelvis, by compressing the pelvic veins acts in the same way. A varicose vein in the leg may ulcerate and cause hæmorrhage: it may also rupture from the mere distension. It is a common thing to find an eczema over the vein, caused by the chronic inflammatory condition which varicose veins always tend to set up.

(2) The *rectum* is a common site for varix, particularly at and just above the anal orifice. The name "**hæmorrhoids**" is applied to varicose veins in this position, and it is usual to speak of **external** and **internal** hæmorrhoids, according as the affected veins are situated, without or within the anal aperture. The veins supplying the lower part of the rectum are in communication with the portal system (through the inferior mesenteric), and with the vena cava (through the internal iliac), but this double outlet does not prevent the frequent occurrence of varix in these lower hæmorrhoidal veins. Hæmorrhoids especially occur when the portal vein is obstructed through disease of the liver. The mucous membrane over them becomes altered in consequence of the continual irritation set up by the passage of fæces, and periodical bleeding takes

place when there happens to be a sudden and abnormal increase of pressure. The veins rupture through the mucous membrane, and the condition is temporarily relieved by the escape of blood. But, in the absence of treatment, the bleeding recurs again and again until the prolonged drain may produce fatal anæmia.

(3) The *œsophagus* at its lower end is sometimes the seat of varix, and fatal hæmorrhage into the stomach has occured from rupture of varicose œsophageal veins.

(4) The *spermatic veins* frequently are the seat of varix. The left, however, is very much oftener affected than the right, probably because the former is the longer of the two, and opens into the renal vein at a right angle. The condition receives the special name of **varicocele.** The varicosity generally begins at the external abdominal ring and extends outwards.

ATHEROMA.

Atheroma in veins is an occurrence of minor importance. It is sometimes set up when veins are subjected to extreme pressure, as in the case of an aneurismal varix. Atheromatous patches are sometimes formed in the pulmonary veins in cases of obstructive disease on the left side of the heart (mitral stenosis especially).

PHLEBITIS.

This is the most common disease which affects veins. Like other inflammations it may occur in acute or chronic form.

There are two **varieties** :—

(1) *Simple* ; (2) *Septic.*

(1) **Simple acute phlebitis** occurs in persons suffering from acute fevers and other degenerative conditions. It may also occur secondary to an attack of fever. In some cases, it arises apparently in a spontaneous manner, and it may be set up by traumatic injury.

Characters.—Under the inflammation the vein speedily dilates, and a thrombus forms in it. " In all cases of acute inflammation of blood vessels, thrombi form in them—the blood coagulating on the inflamed surface." The vein may become obliterated by the thrombus. Phlebitis is very liable to spread, and with it the thrombosis.

Microscopically, the inner coat shows proliferation of the endothelium and connective tissue. The outer coat is partly infiltrated with leucocytes.

Although thrombosis is invariably found in cases of phlebitis, yet we know that the formation of thrombi may be the primary phenomenon, they by their presence inducing inflammation.

(2) **Septic phlebitis.** This occurs as a result of pyæmia, and differs in certain important respects from a simple inflammation. A

thrombus forms in both instances, but in cases of pyæmia this thrombus rapidly softens and breaks down into a puriform mass, having a green colour, and offensive odour. The wall of the vein becomes gangrenous, turning purple and then green. Suppuration also occurs outside the vein, and bacteria can be discovered with the aid of the microscope.

THROMBOSIS.

Thrombosis very frequently affects veins. We have seen that it is a sequel of varix and phlebitis. It will be unnecessary to mention other causes or to dwell on the appearances and changes presented. These have been fully described in Part I.

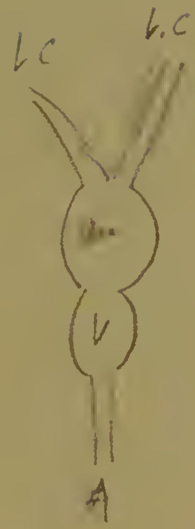
MALPOSITIONS and MALFORMATIONS of the HEART and GREAT VESSELS.

I. MALPOSITIONS.

The heart is very rarely found misplaced to any great degree. When its position is materially altered, there nearly always exists a general malformation of the body. The organ has occasionally been "transposed," *i.e.*, it lies to the right of the sternum with the apex pointing in that direction. This condition is known as **dexiocardia.** It is to be distinguished from that form of displacement which results from emphysema of the lung, where the apex comes to point to the right and the left ventricle is in apposition with the chest wall on the right side of the sternum. In dexiocardia the aorta passes over to the right side, and its branches are correspondingly altered. Usually also, the other viscera in the body are transposed. The heart has occasionally been discovered in the neck, and in the abdominal cavity.

II. MALFORMATIONS.

Malformations of the heart and its great vessels for the most part represent survivals of fœtal conditions. They are due either to arrest of development at certain periods, or to some abnormality—it may be in the blood pressure—which retards the normal cycle of vascular changes during intra-uterine life. That being so, it will be well here to recount briefly the essential facts relating to the **development of the fœtal heart with its vessels.** At a very early period the heart consists of two chambers, an auricle and a ventricle: the auricle receives two venæ cavæ posteriorly, while the ventricle gives origin anteriorly to a single arterial trunk. Each chamber, as well as the artery, subsequently divides into two; and this takes place in each case by the formation of a septum. The ventricular septum is the first to appear, being first evident at the sixth week of intra uterine life. It commences at the apex of the chamber and gradually approaches the base, which it reaches about the ninth week. But its completion at the base is delayed till a late period; and even when entire, the basal part shows a triangular area in which there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is no muscular tissue, and which consists merely of two layers of endocardium with some loose connective tissue between. This area is called the *undefended space*. The common arterial trunk begins to show signs of division as the ventricular septum is approaching the base. A septum passes from each side of the vessel and divides it into two in such a way that one division is connected with each ventricle : that connected with the right ventricle is the pulmonary artery, the other with the left is the aorta. The auricular septum makes its appearance about the eighth or ninth week (*i.e.*, when the ventricular septum is nearly complete). It arises in two portions, anterior and posterior in position. The anterior one appears first and grows backwards, the posterior segment afterwards grows forwards and meets it : complete union, however, does not take place until birth, there being a persistent opening called the foramen ovale.

Now, those malformations of the heart which are compatible with life are usually associated with some anomaly of the *pulmonary artery* or of the *aorta*. Obstruction of either of these vessels leads to imperfect closure of the ventricular septum, the immediate effect of which is, of course, to cause a mixing of the arterial and venous blood. But there are important subsequent results, as will be presently shown.

Pulmonary Stenosis.—The most common **cause** of this condition is disease of the valves, resulting in adhesion of the cusps so as to form a central opening through which the blood is forced with difficulty. This is an instance of fœtal endocarditis, occurring on the right side of the heart (v. page 110). In some cases the orifice of the artery is narrowed, while the valves are normal. Such a condition would appear to result from some disturbance of the blood pressure on the right and left sides of the heart during an early period of fœtal life whereby the arterial septum is displaced, leaving the pulmonary orifice very much smaller than the opening of the aorta. A third form of obstruction is where the artery itself is contracted beyond the valves.

The **effects** of pulmonary stenosis vary according to the time when it takes place. If it occurs at an early period, before the ventricular septum is complete, the blood from the right ventricle as well as from the left will pass into the aorta. This constant passage of blood from the right to the left ventricle will entirely prevent the closure of the septum. In addition, the incomplete septum will be displaced to the left to such an extent that the aorta really arises from the right ventricle as much as from the left. In this way, the heart has virtually but three cavities, one ventricle and two auricles.

But, when stenosis occurs at a more advanced period of intra-uterine life, in fact, when the ventricular septum is almost closed, additional results are obtained. The right ventricle becomes gradually hypertrophied, to compensate for the obstruction to its normal outlet. As a natural consequence of this, there is increased strain thrown on the right auricle; blood accumulates in that chamber, which relieves itself by forcing greater quantities through the foramen ovale; and so the latter is prevented from closing at birth, remaining permanently patent. But we may have a great degree of pulmonary stenosis without any marked patency of the foramen ovale. A third phenomenon which occasionally ensues is a persistent patency of the ductus arteriosus. The lungs being imperfectly supplied with blood through the stenosed pulmonary artery, receive an auxiliary supply after birth from the aorta by way of the ductus arteriosus. (The blood thus passes through the ductus in the inverse direction to that which it takes in the fœtus.)

Pulmonary stenosis sometimes occurs after the ventricular septum is complete. In such a case the chief results are hypertrophy of the right ventricle and patency of the foramen ovale.

Aortic Stenosis. The aorta is occasionally the seat of obstruction during intra-uterine life, though not nearly so frequently as the pulmonary artery. Corresponding results are obtained in each case. Aortic stenosis causes *(a)* patency of the ventricular septum; *(b)* bulging of the septum to the right, causing the pulmonary artery to take origin from both right and left ventricles; *(c)* patency of the ductus arteriosus, allowing blood to pass from the pulmonary artery into the aorta and supply the systemic arteries; *(d)* patency of the foramen ovale. The last two do not necessarily follow.

Children born with any of these malformations of the heart usually suffer from symptoms of respiratory and circulatory embarassment. These symptoms are mainly dyspnœa and lividity, the same as we observe in adults afflicted with valvular disease. The symptoms do not necessarily appear at birth; sometimes they are postponed for months or even years and then apparently develop under the influence of some undue strain on the circulation. The symptoms are usually intermittent to begin with; in the course of time they may become permanent. The face and lips, hands, feet and nails acquire a livid appearance, and this congenital cyanosis is often spoken of as the **morbus cæruleus**. It is due to the venous engorgement consequent on the arterial obstruction.

III. ABNORMALITIES IN THE VALVES.

Congenital malformations are most commonly met with in connection with the sigmoid valves. It has been pointed out that obstruction in the pulmonary artery is usually due to fusion of the semilunar valves so as to form a diaphragm. Sometimes this protrudes into the lumen of the artery in a funnel-shaped form. In some cases two of the segments are fused, so that the valve only possesses two cusps. These changes are no doubt due to fœtal endocarditis, and we can always observe traces of the coalescence of the cusps in the shape of thickenings

along the line of union. Another anomaly, more common in the aortic valve, is the presence of one or more additional segments. Such segments are of small size compared with the normal ones, and the valve is not rendered incompetent by their presence. Occasionally, the margins of the aortic cusps exhibit a fenestrated character, sometimes approaching to the formation of tendinous bands closely resembling chordæ tendineæ. This does not interfere to any extent with the function of the valve, since the fenestration does not extend beyond the lines of contact of the cusps. Again, pouches are sometimes found on the cusps: they are often said to be aneurismal.

ABNORMALITIES IN GREAT VESSELS.

The aorta has been known to arise posteriorly from the right ventricle, and the pulmonary artery anteriorly from the left. The blood from the systemic veins thus enters the systemic arteries without passing through the lungs. One case of this kind survived for three years. The aorta is occasionally double, owing to non-closure of the right arch. Another common malformation is the development of the right instead of the left arch, so that the vessel passes to the right. Sometimes we meet with a congenital narrowing of the aorta, taking place just beyond the origin of the left subclavian. This narrowing has been known in some cases to extend throughout the whole length of the vessel, and to be accompanied by thinning of the wall. This remarkable condition has been found associated with a number of cases of chlorosis, and also interstitial nephritis.

The ductus arteriosus, the fœtal communication between the aorta and pulmonary artery, normally begins to close about the time of birth at the aortic end, and becomes closed after birth towards the pulmonary end. We have seen that stenosis of the pulmonary artery causes the ductus to remain patent after birth. Anything which increases the pressure in the aorta or pulmonary artery tends to cause patency of the ductus, *e.g.*, a congenital narrowing of the isthmus of the aorta will bring it about.

The right subclavian artery sometimes arises from the aortic arch beyond the left subclavian (instead of from the innominate). This necessitates it passing the other branches of the arch from left to right in order to gain the right arm. In this course it may come to press on the œsophagus, and it has been known to do so injuriously—an important point in clinical medicine.

The superior vena cava is sometimes stenosed or entirely obliterated a short distance above its entrance into the heart. In some of these cases the left instead of the right superior cava is persistent. Owing to the course of this vessel it is liable, in the case of the heart undergoing enlargement, to be subjected to pressure by that organ.

DISEASES OF THE RESPIRATORY SYSTEM.

I. The PLEURA.

General considerations.—The pleural sac bears a close affinity to the pericardial sac in its structure, and in many of its relations. But the intimate connection which the pleura has with the lung renders it liable to be implicated in many of the diseases of the lung; and further, the fact that the pleural and peritoneal cavities are in continuity by means of lymphatic vessels (which pierce the diaphragm) allows of secondary processes arising in either sac by direct extension from the other.

HYDROTHORAX.

This means a distension of the pleural sac with serous fluid, in other words a dropsy of the pleura. It usually occurs as a part of general dropsy, the result of mitral disease for example, hence both right and left pleuræ are involved. Malignant growths may give rise to hydrothorax. Cancer, especially when it involves the pleura, often causes a dropsical effusion; perhaps the growth obstructs the lymphatics, thus entailing deficient absorption of the normal lubricating fluid. The effect of hydrothorax depends upon the amount of the effusion. If the effusion is considerable it may so compress the lung as to cause collapse. Sometimes it happens that one pleura has been adherent from an old pleurisy; in such a case, the serum instead of distending the pleural sac effuses into the lung tissue, and causes œdema of the lung.

HÆMORRHAGES.

Ecchymoses are found in the deep layer of the pleura after death from asphyxia.

Larger hæmorrhages are uncommon. They may follow traumatic injury, or result from rupture of an aortic aneurism into the cavity. Occasionally a pleuritic effusion is mixed with blood, constituting a "hæmorrhagic pleurisy." This is usually associated with the presence of some new growth in the pleura.

PLEURISY,

Or inflammation of the pleura, is a very common disease.

Its **causes** are numerous, and may be grouped shortly thus:—(1) Idiopathic. The cause is frequently undiscoverable. Very often the disease is attributed to "a cold." (2) Extension of inflammation from neighbouring parts, vizt., pericardium and myocardium, mediastinum, and peritoneum. (3) Morbid conditions of the blood,—acute rheumatism and especially Bright's disease. (4) Previous disease of the pleura,—cancer and tubercle. (5) Traumatic, from injury of chest wall, *e.g.*, a fractured rib.

Characters.—The disease may be *acute* or *chronic*. An acute spontaneous pleurisy is nearly always limited to one or other pleura, *i.e.*, it is "unilateral"; and it commences as a rule over the lower and lateral part of the lung, probably because the chest wall affords least protection from external influences at that part. The process of inflammation in its anatomical details is closely analogous to that in the pericardium, and so the student is referred back to acute pericarditis for a description of the appearances. It will be sufficient to state here that the process may be divided into three stages,—(1) congestion; (2) serous effusion and deposit of plastic lymph on the inflamed surface; (3) absorption of fluid and organization of lymph.

The inflammation may take the form either of a "dry" or "serous" pleurisy, according to the extent of the effusion. The serous effusion is a limpid straw coloured fluid, which does not spontaneously coagulate.

Results.—In favourable cases, the inflammation subsides without producing any organic change, and the fluid is absorbed. Such a perfect return to normal, however, is not common. As a rule, more or less adhesion occurs between the visceral and parietal layers, and persists during the remainder of life. This does not necessarily interfere with the due expansion of the lung, but frequently the adhesion is so extensive as to bind down the lung in great measure, and so impede the function of the organ. The effusion in a serous pleurisy may reach an extraordinary degree without permanently damaging the lung, though a point is often reached at which the lung can no longer resist the increasing pressure, and collapse is the result. The collapse is more often partial than complete. Sometimes the fluid, when the effusion has persisted for a length of time, thickens and caseates, subsequently becoming calcified; and after death we find calcareous plates in the pleura. Again, the fluid occasionally becomes purulent, and then the condition is spoken of as "empyema." This only happens in very intense inflammations. Lastly, an acute pleurisy often becomes chronic.

Varieties.—I. **Empyema** may be defined as a purulent pleurisy. We have seen that it occasionally follows a simple pleurisy: this is especially the case in children. It may be accepted as an axiom that purulent pleurisy is the rule in young children, while in adults it is the exception. Many conditions of the lung give rise to it, for example, lobar pneumonia, abscess, infarction and tuberculosis. Then it may be set up by traumatic injury, such as a gun-shot wound. Occasionally, an abscess of the liver has perforated the diaphragm and opened into the pleural sac, setting up a suppurative inflammation of the pleura. But, in very many cases, empyema originates as a primary and acute condition, its causation being unknown. An acute empyema generally involves the whole pleura. In other cases, the condition is commonly localized, and the so-called "loculated empyema" is especially found over the lower lateral aspect of the lung. Sometimes it occurs between two lobes, when it is spoken of as "inter-lobar," or between the diaphragm and base of the lung. The disease is usually unilateral, unless it has originated in some constitutional disorder.

The **morbid phenomena** and sequelæ vary with the acuteness of the disease as well as with its cause. When the suppuration has come on in the course of a simple pleurisy, the process is prolonged and leads to great thickening of the pleura. The membrane shows a shaggy surface, studded with vascular granulations, and resembles the wall of any ordinary abscess. Between the layers there is a quantity of pus, often thick, but usually "laudable." This either becomes absorbed, so allowing of partial adhesion between the two surfaces, or is transformed into caseous material. A natural cure thus results; but very frequently this result has to be promoted by evacuating the pus and so hastening the union of the visceral and parietal pleura. In a localized empyema, which is usually chronic, the same phenomena are observed in a limited area. The pus is frequently very thick and curdy, and contains shreds of tissue. The process usually terminates by caseation and calcification of the pus, and adhesion of the pleura. An acute empyema generally runs a rapid course and ends fatally unless surgical interference be resorted to. On examining the pleura after death, it shows but little thickening, while the quantity of pus present in the pleural cavity may be very great. Unless in traumatic cases, the pus found in empyema is generally laudable, though the micrococci of suppuration are abundantly found in it.

When an empyema does not undergo a natural cure, the pus sometimes burrows in various directions until it procures a free exit. Thus, it is a common enough thing for the chest wall to be perforated in one of the intercostal spaces. Another result, also fairly common, is an extension of the suppuration into the lung, and the establishment of a fistula between the pleural cavity and a bronchus. Through this the contents of the pleural sac may be partly evacuated by the act of coughing. Occasionally the diaphragm is perforated, and the pus is poured into the abdomen,—this is always fatal. Cases have even been recorded of the pus making its appearance externally in the region of the buttocks, and even in the popliteal space.

II. Chronic Dry Pleurisy is a very frequent result of disease of the lung, and terminates as a rule in localized adhesions. Such adhesions can be very constantly detected in the *post mortem* room in elderly subjects. In cases of phthisis, for instance, this is almost always observed The adhesions often assume the form of flat firm laminated plates, resembling the "flat fibromas" found on the capsule of such organs as the spleen.

III. **Hæmorrhagic Pleurisy.**—In this condition, the inflammatory effusion contains blood. It is very rare in a simple pleurisy, usually there is some new growth that predisposes to it. Of these, cancer is the commonest, and so the condition is often synonymous with "cancerous pleurisy." In addition, however, diseases of the lung, which lead to obstruction of the pulmonary veins, may cause a hæmorrhagic pleurisy; interstitial inflammations are of this class.

IV. **Syphilitic Pleurisy.**—Evidences of inflammation are occasionally seen on the pleura in the case of persons who die tainted with constitutional syphilis. These are in the form of wrinkles and cicatrices of the pleura, often attended with thickening of the membrane; and their usual site is over the lower part of the lung on its posterior aspect.

V. **Tubercular Pleurisy.**—The presence of tubercle in the pleura frequently gives rise to inflammation of the membrane. It should be noted that the inflammation may lead to a simple serous effusion, although this is not often the case; when fluid is effused it is usually thick, and becomes caseous. Frequently a dry pleurisy is set up, resulting in the formation of adhesions. When the inflammation is chronic (and adhesions consequently form), typical giant cell systems can be found in the adhesions. In acute forms, and those attended with rapid caseation, giant cell systems are few in number, and are limited to the deeper part of the membrane.

TUBERCULOSIS.

The pleura is very often affected with tubercle secondarily to the lung. Possibly the disease sometimes affects the pleura primarily, but such instances must be rare. It is known, however, that in cows the pleura is frequently a seat of tubercle when the lung is unaffected.

Characters.—One of two conditions may be present. On the one hand minute grey granulations are scattered over the surface of the pleura; or, large caseous nodules occur here and there. In exceptional cases, the pleural cavity becomes filled up with thick fungating masses, a condition characteristic of the bovine affection. These masses become caseous, and may calcify. The pleura is usually more or less inflamed.

TUMOURS.

Secondary growths are common in the pleura. They usually arise by direct extension from the mediastinum or peritoneum. Various forms of **sarcoma** are met with, principally **lympho-sarcoma** and **melanotic sarcoma**. **Cancer** also frequently occurs; and the lung is nearly always involved along with the pleura. Cancer of the peritoneum often extends to the pleura. A cancer of the mamma may infiltrate the chest wall and extend to the pleura. Tumours of the pleura are usually in the form of flat, soft masses, usually sessile, but occasionally pedunculated and polypoid. Since cancers infect by way of the lymphatics, it often happens that the growth marks out areas on the pleura, which indicate the course of the lymphatic vessels around the margins of the lobules.

It has been pointed out that tumours give rise to inflammation of the pleura. A simple dropsical exudation often accompanies cancerous formation.

II. The LUNG.

COLLAPSE.

This is a term employed to indicate a condition of the lung (or part of that organ) in which the air cells and finer bronchi are no longer expanded, so that they contain no air. Thus the organ resembles the fœtal lung before inflation, the internal surfaces of the alveoli being applied to one another. There are several forms of collapse, and a primary classification into *congenital* and *acquired* collapse will be found useful. The former is technically spoken of as "atelectasis" (non-expansion), while the latter receives the name of "apneumatosis" (deprival of air).

ATELECTASIS.

This is commonly enough found in new born children, whose lungs have never fully expanded. It is usually the result of simple weakness, such children being nearly always puny and feeble, and unable to inspire with sufficient vigour to inflate the entire lung.

Sites.—Generally some peripheral part of the lung suffers, and especially the lower lobe towards the root and base.

Morbid Anatomy.—In the above position we find a portion of the lung depressed below the surface and redder than the surrounding expanded parts. It is further distinguished from the latter by being firm to touch and non-crepitant. If examined carefully, it will be found to consist of a number of lobules, and its shape will probably indicate that it is the district supplied by one or more bronchi. If air be blown into the lung through any of these bronchi, the atelectatic portion can be made to expand.

Sometimes the greater part of a lobe is involved, and even an entire lobe may be found airless. Should the infant survive, the lung will probably expand in time; but if the atelectasis persists long, expansion may become impossible.

APNEUMATOSIS.

Acquired collapse of the lung occurs from a variety of causes, and assumes characters which vary with these causes. So it is convenient to discuss the causes along with the form of collapse which they give rise to. But it may be broadly laid down that collapse of the lung is brought about by the influence of any agency which renders the pressure on the external surface of the lung equal to that exerted by the atmosphere from within.

I. TOTAL COLLAPSE.

This name signifies collapse of an entire lung, or of a lobe, or other massive portion of a lung.

Causes.—(1) Puncture of chest wall, by a knife stab for instance. (2) Rupture of the surface of the lung, *e.g.*, the result of a broken rib. (3) Serous pleurisy is the most common cause of collapse. The effusion, gradually accumulating, exerts a constant pressure upon the lung and expels the air out of it. (4) Obstruction of either bronchus by a foreign body may cause collapse of an entire lung. The left bronchus, on account of its direction, is much more liable to be plugged by a foreign body passing down the trachea than the right. How such an obstruction can be the means of bringing about collapse will be explained in connection with the "mechanism of collapse."

Morbid Phenomena.—In the case of pulmonary collapse following a serous pleurisy, the lung is steadily compressed upwards and backwards until it comes to form a solid fleshy mass applied to the inner and back part of the thoracic wall. This subsequently atrophies to a large extent, the organ being now functionless. The appearance of the lung tissue, both naked eye and microscopic, is very similar to that found in cases of partial collapse, so the description of the latter will apply to this (see next page).

When an entire lung collapses in this manner, the other lung usually exhibits some degree of compensatory emphysema, and gets into a condition of chronic congestion.

II. PARTIAL COLLAPSE.

The most common kind of collapse met with as an acquired condition is that which affects limited portions of the lung. These portions may represent distinct and entire lobules, when we speak of **lobular collapse**: or they may be undefined areas of lung tissue situated irregularly in the organ, and when that obtains we apply to it the name of **disseminated** or **scattered collapse**. It is convenient to consider these two forms together.

Causation.—Partial collapse of the lung generally depends on a combination of two factors: (1) Defective respiratory force, and (2) Obstruction to the entrance of air into the lungs.

The first of these arises from some impediment to the free movement of the thoracic parietes. Thus *(a)* in the children's disease—rickets—the ribs with their cartilages are softened, so that they can neither afford adequate purchase to the respiratory muscles nor sufficiently resist external pressure. *(b)* Disease of the intercostal or other respiratory nerves sometimes renders the muscles useless or feeble. *(c)* Certain diseases, fevers especially, often weaken the patient and embarrass his respiratory efforts. *(d)* Various mechanical obstacles may impede the free movements of the chest. This has been experienced in cases of abdominal tumours or ascites causing great upward distension of the diaphragm, in extensive enlargement of the heart or distension of the pericardium with fluid, in large aneurisms and in thoracic tumours. The careless bandaging of the chest in surgical practice has occasionally been the means of producing a similar effect.

(2) Air may be prevented from entering the lungs in many ways, and the obstacle to its entrance may be situated in any region of the respiratory tract. Thus, obstructions have occurred in the larynx, trachea, main bronchi, and various parts of their many divisions. The commonest sort of obstruction is in the shape of a foreign body accidentally admitted through the glottis, but an aneurism or tumour frequently exerts its pressure on one or other of these passages, narrowing their lumen and impeding the free passage of air through them. In the smaller bronchi and terminal bronchioles the obstruction may be produced by bronchitis, and this is generally the case in children. The swelling of the mucous membrane combined with the hyper-secretion of mucus, plugs the bronchioles and cuts off the supply of air from their corresponding alveoli. Lobular collapse is usually a sequel of bronchitis. The exudation or false membrane which sometimes extends from the trachea to the minute bronchioles in diphtheria, has a similar effect. The inhalation of blood into the bronchioles, consequent upon hæmorhages into the air passages, has frequently led to a like result.

Sites.—Where there is a general obstruction to the entrance of air, the lung undergoes collapse at certain definite positions, one region after another becoming affected in consecutive order. This is particularly obvious when the obstruction is associated with weakened inspiratory effort. Under such circumstances, then, collapse is most apt to occur first at the lower margins of the lower lobes of both lungs. Secondly, it occurs in the tongue-shaped process of the upper lobe of the left lung. Next, it appears in the middle lobe of the right lung, towards its anterior part. And next, the collapse affects the posterior surfaces of each lung, involving all the lobes.

In cases of *fever*, where death is preceded by great exhaustion, one commonly finds collapse present in the lower and posterior parts of each lung. These are the least movable portions of the organ.

Lobular collapse, following bronchitis, may occur at any part of the lung—in fact, wherever the bronchitis is most intense—but it is usually most obvious at the base and posterior border of the lung, or along the free margins.

Morbid Phenomena.—In the condition of disseminated collapse, the collapsed areas are detected by being depressed below the level of the surrounding lung tissue. They are tolerably well defined, their margin having an irregular angular outline owing to the circumstance

that it corresponds to the edges of certain lobules. The collapsed tissue is dry and smooth, and since it contains no air does not crepitate on pressure. It is usually of a dark blue colour, both on surface and on section; this appearance is due to the absence of blood in the part, and close aggregation of the particles of carbon pigment which normally are scattered over the organ. When dropped into water the tissue sinks, so solid has it become. In an early stage of the condition, the affected parts are found congested, but latterly they become anæmic. Their function being now in abeyance, the collapsed parts atrophy to a large extent.

Lobular collapse is not so readily detected. The collapsed lobules are represented by small brownish spots, depressed below the surface, and often further characterized by being surrounded by an emphysematous zone of lung tissue. The lobules are usually in a state of congestion.

Microscopically.—In a portion of old collapsed pulmonary tissue, the structure of the lung is seen to be in great degree obliterated. Everything is crowded together and atrophied. No air cells can be distinguished, their position being indicated perhaps by some remnants of epithelial cells, the debris of their walls. The capillary vessels undergo obliterative endarteritis and disappear. The only elements which persist in or near the collapsed parts are the larger bronchi and vessels.

It is very difficult to distinguish between atelectasis and apneumatosis. As a rule the lung tissue in the former case is redder and more fleshy than in the latter, and the pulmonary epithelium may be persistent and fairly normal.

MECHANISM OF COLLAPSE.

When collapse is due to the presence of air or fluid in the pleural sac, it is quite easy to trace cause and effect here. The pleural cavity being practically a vacuum under normal circumstances, the lung expands during inspiration with every expansion of the chest wall, and by virtue of its elasticity recedes when the chest falls during expiration. But whenever this vacuum is ~~distributed~~ dis to any extent in the manner mentioned, the lung suffers to a corresponding degree, and some portion undergoes collapse since the entire lung cannot expand. In the case of a communication being established between the pleural cavity and the external air (whether by a wound through the chest wall or by a rupture at some peripheral part of the lung), it will be understood that the internal atmospheric pressure is now equalized by the external pressure; and the organ as a matter of course, by virtue of its elastic nature, contracts or in other words collapses.

But something very different from this theory is required to explain how a lung collapses when an obstruction exists in some part of the respiratory tract. Suppose a foreign body has become immovably fixed in one of the larger bronchi and absolutely prevents the passage of air along the bronchus in either direction—into the lung or out of the lung. The consequence is that the air which is confined in the portion of lung supplied by the bronchus becomes gradually absorbed, while the lung simultaneously contracts. The absorption is further promoted by the elastic pressure exerted by the lung tissue, and so the air is partly absorbed and partly expelled. This double process soon results in collapse of that portion of the organ involved. It has been proved that this absorption of the constituent gases of the air does actually occur; the oxygen is the first to disappear, next the carbonic acid, and lastly the nitrogen.

There is another hypothetical mode whereby a foreign body lodged in a bronchus is the means of bringing about collapse of the district supplied by that bronchus. This is said to occur specially in the smaller bronchi, particularly in cases of bronchitis. A plug of mucus is supposed to lodge at the bifurcation of a bronchus, and in such a manner that it moves to and fro in the bronchus with every act of inspiration and expiration: it thus causes a partial obstruction, and performs a similar function to the valve of an air pump. When an expiration is made the outgoing air carries the plug before it a certain distance and the lung is partly emptied. At the commencement of inspiration the plug is carried back to the bifurcation, and prevents in great measure the air from entering the two divisions of the bronchus. The repetition of this process a number of times results in the air being entirely pumped out of the involved piece of lung and so it necessarily collapses.

EMPHYSEMA.

Under this name are comprised two different and distinct conditions. One of these may be described as the converse of collapse, since it consists essentially in an over distention of the air cavities of the lung, and is spoken of as *common* or *vesicular emphysema*. In the other, which may be compared with the surgical condition of "cutaneous emphysema," we have to deal with an escape of air from the pulmonary air cells into spaces formed in the interstitial and surrounding connective tissue. To this condition the synonymous terms of *interstitial* and *interlobular* are indifferently applied.

I. VESICULAR EMPHYSEMA.

Definition.—A permanent over distention of the air-containing cavities of the lung and the persistent effects of this upon the lung structure.

Causes.—(1) **Exciting.** Emphysema is produced by some force which, frequently repeated, causes an abnormal inflation of the air vesicles of the lung. This force is to be found in the constantly acting "ſea-valve"

respiratory mechanism. In the first place, the act of *inspiration* may under certain circumstances, lead to the production of emphysema. Thus, the latter may ensue when, during the phase of inspiration, a section of the pulmonary area in the thorax remains unoccupied by the lung. Such a state of matters obtains if a portion of the lung (from whatever cause) does not expand along with the chest wall: the vacuum thus created must be filled up either by a falling in of the chest wall over the region or by some adjacent part of the lung becoming permanently distended, *i.e.*, emphysematous. It is in this way that a lobule of the lung which is in a state of collapse so often comes to be surrounded by a zone of emphysema; it is observed very constantly after bronchitis. Adhesions of the lung often lead to a similar result; the surrounding non-adherent tissue becomes emphysematous. So with cicatrices in phthisis. It may also occur where portions of the lung become condensed as a result of lobular pneumonia for instance. Emphysema occurring under any of these conditions is spoken of as being *compensatory*.

In the second place, violent acts of *expiration*, performed with the glottis closed, are a common source of emphysema. In the performance of such efforts, the lungs are compressed by the action of the diaphragm and the various muscles on the chest wall, and since the pressure so exerted must be equally diffused over the pulmonary tissue, it follows that those parts of the organ which derive least support from the thoracic parietes will suffer from the increased pressure, and exhibit the effects of emphysema, if that process does occur. What those parts are will presently be mentioned. Now, that emphysema does occur in this manner is proved by the fact that it is frequently observed in those who have been in the habit of playing wind instruments. But it is very commonly the result of the excessive effort put forth in the act of coughing, so we should expect to find emphysema occurring as a sequel to these diseases of which coughing is a symptom. And "it is met with pre-eminently in bronchitis."

(2) **Predisposing.** The occurrence of emphysema is always aided by defective nutrition in the lung tissue, whereby its elasticity is impaired. Senile changes of an atrophic nature very commonly involve the lung tissue and so are a fruitful source of such impairment. In the chronic bronchitis of old persons, this primary atrophy is doubtless an important predisposing cause of emphysema, so much so as to lead to the use of the term "senile" or "atrophous" emphysema. Chronic wasting diseases also naturally predispose to the condition. Lead poisoning is another recognized cause. Rigidity of the chest wall apparently favours the condition, when the chest tends to assume the attitude of full inspiration.

Although one generally finds some predisposition to account for the diminished resisting power of the air cells, yet it should be borne in mind that the mere over-distension of these air cells has a natural tendency to impair their elasticity, and so it cannot always be determined to what extent the condition may owe its origin to the one cause and how much to the other.

Sites.—Emphysema has been observed in very varying situations in the lung. The locality where we meet with compensatory emphysema will of course depend on the situation of the lesion which gives rise to it. But in the more common and important form of emphysema arising from bronchitis and other kindred pulmonary diseases, one invariably looks for the evidence of the condition at those portions of the lung which are known to be least adequately supported by the thoracic parietes, and where consequently there is least resistance to their expansion. These portions are in order (1) the anterior margin, (2) the base, especially at its margins, and (3) the apex.

The left lung is commonly more emphysematous than the right, a fact to be explained by the assumption that it is less perfectly supported than the right.

Morbid anatomy.—The first thing that strikes one on opening the thoracic cavity is that the affected lungs are more voluminous than normal; this is specially noticeable in front. But they are usually found to be deficient in weight. In the situations above enumerated, the organ is abnormally pale, and exhibits a series of rounded elevations known as "emphysematous bullæ." They are really cysts containing air. Under the fingers these feel "like a mass of feathers in a silk bag," being somewhat harsh to touch and elastic. On squeezing one firmly the contained air can be forced into an adjacent bulla, and if any one of the bullæ be punctured with a knife, the air can be expelled from the whole series through the single opening. A faint crackling sound accompanies the passage of the air from one bulla to another. On section, the lung feels dry and harsh, the tissue appears coarser than natural, and the air spaces are evidently much enlarged.

Microscopically.—The distensile force by its mere mechanical effect produces extensive alterations in those portions of the lung tissue on which the force tells. The infundibulum with its surrounding cluster of air cells suffers first, undergoing gradual and progressive dilatation. This process imparts a globular shape to the infundibulum, and stretches the alveoli so that they become flattened and exhibit an enlarged aperture of communication with the former. Soon the wall between adjacent alveoli atrophies, and when this atrophy has gone on to its full extent the alveoli become practically obliterated, and can only be distinguished by the presence of minute projections attached to the wall of the now enormously inflated infundibulum, and which are the remains of the alveolar septa. The alveolar passages are in their turn distended and assist in the formation of these cavities. Since the cavities are

continually pressing one against another, this mutual pressure necessarily produces atrophy of their walls, and in such a way the air containing cysts or "bullæ" are eventually produced. Naturally, the great distension which certain of the infundibula undergo in the course of development into these cysts must necessitate the atrophy and complete disappearance of many others; consequently at some parts nothing is to be seen but a collection of bullæ.

In addition to these phenomena, the result of mechanical agencies, we must note certain other phenomena associated with the nutritive alterations in the pulmonary tissue. One of the earliest processes that takes place in the production of emphysema is the obliteration of capillaries in the aveolar septa. This appears to result largely from the mechanical pressure, aided by the stretching of the vessels over the expanded air-cells; it has also been attributed to thrombosis. But, however induced, it necessarily implies imperfect nutrition in the areas involved. In the larger branches of the pulmonary artery, atheroma can be detected sometimes. Following the disappearance of vessels, the epithelium of the air-cells undergoes fatty degeneration, then atrophies and is absorbed. The elastic element is subjected to the same process, consequently the lung loses its normal tendency to collapse, and hence the reason why the affected parts remain distended when the lung is removed from the thorax. The wall of the air-cell thus gradually disappears, leaving but scant traces of its existence.

At certain parts, additional changes are observed. The openings of the infundibula are palpably thickened, and on examination this proves to be due to an overgrowth of muscular fibre at that particular part. The overgrowth is probably of an irritative nature, resulting from inflammatory reaction at an early stage.

Effects.—As a result of the enlargement of the lungs the chest capacity becomes permanently increased, the chest wall assuming the attitude of full inspiration. In advanced cases the chest becomes "barrel shaped," the sternum being protruded forwards, while the transverse diameter of the cavity is diminished. The heart is displaced downwards and to the right, the diaphragm descending with it. The descent of the diaphragm induces on the right side depression of the liver. Since the vascular area in the lung is much diminished by the obstruction and obliteration of many of the pulmonary vessels, the right ventricle is called on to perform additional work in forcing an equal amount of blood through a smaller number of vessels. This causes that chamber to hypertrophy in order to compensate matters; and very commonly dilatation occurs at the same time, for the ventricle is often inadequate to the task of making full compensation. This latter circumstance can often be accounted for by the fact that the heart is imperfectly nourished, and becomes fatty and consequently weak.

Following upon this, all the effects of venous engorgement may ensue, a sequel which often proves fatal.

II. INTERSTITIAL or INTERLOBULAR EMPHYSEMA.

This condition is characterized by the presence of air in the meshes of the connective tissue which supports the lung structure. It is not a common condition, and it generally occurs in vesicular emphysema from rupture of the distended bullæ. This results in the production of small air bubbles situated on the surface (immediately beneath the pleura) and marking out the lobules. The air sometimes makes its way along the connective tissue spaces between the lobules, and has been known to reach the root of the lung. The bubbles occasionally burst, and pneumothorax supervenes.

Interstitial emphysema sometimes results from injury. It has also been caused by violent expiratory efforts with the chest wall fixed; thus it has been found after death from whooping-cough and diphtheria.

ANÆMIA

Of the lungs is not usually a marked pathological condition. In cases of death from hæmorrhage, the lungs, in common with other internal organs, exhibit more or less pallor: it is to be noted, however, that in certain cases the lungs are not anæmic, but, on the other hand, are œdematous and congested (*v.* page 84).

ŒDEMA,

Or dropsy of the lungs, consists in the presence of blood serum in the air-cells of the organ. It is brought about by the same factors which govern the production of dropsical effusion in other parts. After many chronic debilitating diseases, it is very common to discover some œdema of the lungs post mortem. This is to be attributed to weakening of the heart, combined with a deterioration of the blood, and is no doubt produced shortly before death. But œdema of the lungs also occurs as a symptom in the course of anæmia and Bright's disease. And it is of frequent occurrence in cases of severe heart disease, when there is generally coincident venous congestion: such instances are to be explained by a lack of propulsive force in the left ventricle, whereby the arterial return from the lungs is embarrassed, while the right ventricle continues to pump its normal sufficiency of blood through the pulmonary artery.

Distribution.—The œdema is usually found in the lower and posterior parts of the lung, but in renal dropsy the apex is often affected in addition. It is always more marked when the pleura is adherent, a circumstance which probably indicates that the condition is aided by

anything which impedes free respiratory movement (*vide* "Hydrothorax," page 141).

Characters.—The lung is generally pale and watery, airless, heavier than normal, but does not sink in water. On section, the pulmonary tissue appears denser than natural, and on squeezing it a quantity of thin frothy fluid exudes from the air-cells. When the dropsy is of cardiac origin the blood-vessels commonly show congestion. **Microscopically**, nothing characteristic is observed. The epithelium of the air-cells exhibits some swelling.

CHRONIC VENOUS CONGESTION.

This results from obstructive disease of the left side of the heart, chiefly mitral stenosis or incompetence of long standing. But it often occurs in connection with aortic disease.

Morbid Phenomena.—These vary with the duration of the exciting factor. In cases of recent origin, the lungs are found to be abnormally voluminous. They are of a reddish brown colour, especially posteriorly, and on making a section a quantity of dirty red fluid exudes. The organ may be slightly emphysematous, and the bronchi show signs of inflammation. In prolonged cases the lung acquires characters which originally led Virchow to adopt the name of **brown induration** in describing the condition. The colour of the organ superficially is a reddish purple, and the inter-lobular septa are very conspicuous on account of their being deeply pigmented. At the free borders of the lung there is commonly some emphysema, and at those parts also one observes a number of more deeply coloured patches, very firm to touch, and slightly raised above the surface. A large quantity of dirty red fluid, frothy from admixture of air, exudes when a section is made. The patches at the surface are then seen to be wedge shaped; if a portion of one be dropped into water it sinks, being of solid consistence. The surface of section presents a characteristic dull brick or rusty red colour, and is moist and somewhat firm to touch. At several points, particularly in the lower and posterior portion of the organ, patches of tissue are detected which vary in size from half-an-inch to an inch, though showing no sharp outline, and are firmer and drier than the general surface. They are somewhat harsh also, and cut with a gritty feel: when squeezed a rusty brown fluid exudes. The inter-lobular septa are deeply pigmented, likewise the bronchial glands; and both these structures are in a state of inflammatory hypertrophy. On examining the bronchi they are found to be in a state of chronic bronchitis; this condition is one of the most persistent secondary effects of chronic pulmonary congestion.

Microscopically.—The wedge-shaped masses at the surface of the lung prove to be haemorrhagic infarcts (*v.* page 21) being composed mainly of a mass of blood corpuscles. This so called "pulmonary apoplexy" is very frequently associated with venous congestion of the lung. The other changes in the lung are a sequel to dilatation of the blood-vessels with its attendant results. The capillary vessels in the walls of the alveoli enlarge and become tortuous, often forming varicose loops which project into the air cell. The persistent pressure causes many of these vessels to rupture and blood is extravasated into the air cells: such hæmorrhages occur repeatedly. As a result of the presence of the solid elements of the blood, the alveolar epithelium proliferates; the epithelial cells absorb the red corpuscles; these break down in the interior of the cells and liberate their colouring matter which appears in the form of golden brown pigment granules. These pigment containing cells pass into the lymphatic system, are conveyed along the perivascular and peribronchial lymphatics, in which they lodge; large numbers of them are deposited in the deep layer of the pleura, and in the bronchial glands. In whatever position these cells lodge, the pigment which they contain appears to exert an irritative effect, producing fibroid thickening of the connective tissue structure. This naturally renders the lung very firm in consistence. In the isolated firmer patches which are scattered through the organ, the changes in the air cells are seen to most advantage If the exudation be examined under a high power (× 300) it will be found to consist of granules of pigment, large flattened cells in which are numerous similar granules, and great numbers of degenerated red blood cells.

Associated conditions.—The frequent occurrence of emphysema and pulmonary apoplexy has been mentioned. The bronchitis which often co-exists with passive hyperæmia may be regarded as a direct effect of the latter. The obstruction to the pulmonary circulation attendant on the congestion leads to hypertrophy and dilatation of the right ventricle.

HÆMORRHAGIC INFARCTION

Of the lung, or "pulmonary apoplexy," frequently occurs side by side with chronic venous congestion. It generally results from embolism of the pulmonary artery, the embolus lodging in one of its branches. But such embolic plugging does not invariably cause infarction, because the pulmonary capillaries are comparatively large and anastomose very freely, and besides the lung tissue is nourished by the bronchial arteries. These facts determine the site of an infarction. If the portion of tissue supplied by the obstructed artery be situated in the midst of lung tissue whose capillaries are filled with actively moving blood, then the circulation in the affected portion is maintained, and infarction does not take place. As a matter of experience we find that hæmorrhagic infarction rarely occurs in the central parts of the lung. But if the haemorrhagic infarcts

district supplied by the plugged artery is situated at the periphery, and especially at such a position as the margin, where it is to a large extent bounded by the pleura, then circumstances favour the occurrence of infarction. And so it is that hæmorrhagic infarction of the lung usually has its seat at a margin of the organ, particularly at the edge of a lobe. Generally, the ~~phenomenon~~ occurs just about the point of death. phen

Characters.—These have been before described in a general way (*v.* page 21). The infarct as a rule is red in colour, and is not encapsuled like those found in other organs. Necrotic changes never supervene in the infarcted area, by reason of the peculiarites in the vascular arrangements above mentioned.

Microscopically.—The infarct is seen frequently to correspond with the distribution of a bronchus. Within the alveoli, there is an enormous aggregation of red blood corpuscles, and scattered here and there are a few catarhal cells derived from proliferation of the epithelium. The corresponding pulmonary vessels contain thrombi, that in the artery being light red while that in the vein is dark.

Results.—In a few instances the traces of infarction may possibly disappear and the circulation be re-established. In a number of cases, the infarcted area shrinks and remains indurated and pigmented. In rare cases a localized pleurisy is set up over the infarct, and still more rarely suppuration occurs, resulting in abscess formation.

EMBOLISM OF PULMONARY ARTERY.

Plugging of the pulmonary artery by a blood-clot has been referred to in relation to infarction. There is another form of embolism which is prone to occur in the lungs, viz., **fat embolism.** The conditions under which this occurs will be found enumerated on page 22.

Characters.—The phenomena to be observed with the naked eye are these:—(1) Scattered under the pleura are a number of minute ecchymoses; (2) the lung is œdematous in the lower lobe; (3) patches of "broncho-pneumonia" are present throughout the lung. **Microscopically,** collections of fat droplets are found occupying the finer branches of the pulmonary artery and extending into the capillaries.

PULMONARY HÆMORRHAGE AND ITS CAUSES.

We have already seen that hæmorrhage takes place into the lung in chronic venous congestion, pulmonary apoplexy, and fat embolism. It also occurs as a result of injury, and of rupture of aneurisms on the pulmonary artery. We have yet to learn that it also occurs in pneumonia and phthisis.

Hæmorrhages not unfrequently occur in the lung in the course of septic fevers, malignant disease, and anthrax. In addition, they may occur in such diseases as purpura hæmorrhagica and pernicious anæmia, when one finds hæmorrhages in other organs.

PNEUMONIA.

This signifies an inflammation of the lung. On account of the complex nature of its structure, there is a great variety of inflammatory processes in the lung, and these may be primarily grouped into three main types :—

I. Croupous or Fibrinous Pneumonias, analagous to the inflammation of a serous surface, and characterized by exudation of coagulable lymph into the alveoli.

II. Catarrhal Pneumonias, in which there are catarrhal changes in the air cells, as well as in the mucous membrane of the bronchi.

III. Interstitial Pneumonias, involving especially the connective tissue structure of the lung.

I. ACUTE CROUPOUS or "LOBAR" PNEUMONIA.

This is an acute febrile disease, and is that common affection which is popularly spoken of as "inflammation of the lungs."

Causation.—The particular irritant or irritants which set up this condition have not been determined. But it seems certain that the exciting agent is conveyed to the lung by the blood. Acute pneumonia has been shown to follow the introduction into the blood of various chemical irritants, carbolic acid for instance. In most instances it apparently results from undue exposure to cold and wet, but such cases are now being explained by the theory of organismal agency. Koch has discovered the presence of bacteria, and states that they always exist in those parts of the lung where the disease is active. Although the inflammation is usually endemic, yet it sometimes becomes epidemic, a fact which favours the zymotic theory.

Morbid Anatomy.—The inflammatory process is usually described as occurring in four stages ; this is very convenient for descriptive purposes, but it must be borne in mind that these stages run insensibly one into the other, forming a continuous chain of events in one process. Further, one is not always able to trace the inflammation to its ultimate end, because it very often happens that the process is arrested during the second stage, and indeed in the majority of cases the condition of "grey hepatization" in the third stage is not reached.

The inflammation may attack only one lung, but it often involves both—either simultaneously or in succession. It nearly always commences at a definite point, usually the base ; and it spreads upward by direct continuity. From the nature of its progress the disease is usually manifested in more than one stage simultaneously in the same organ.

Thus it is common to find the lower lobe well advanced in the second stage, while the upper lobe exhibits the phenomena of the first stage. This can be demonstrated by making a vertical section of the lung from apex to base.

We shall now consider the stages as they occur in order :—

(1) ACTIVE CONGESTION.

On examining the lung with the **naked eye** it is seen that the lower lobe, and perhaps a portion of the upper lobe, is swollen and of a dark red colour. To the touch it is inelastic and the tissue pits on pressure. It is heavier than natural, but does not sink in water for it is not altogether airless; crepitation can still be felt, though in small degree. The pleura over the affected area shows marked hyperæmia, but there is an absence of pleurisy. On section, the surface of the lower lobe appears of a bright red colour and is evidently œdematous. On squeezing the lung a reddish serum flows from the air cells, carrying bubbles of air with it; and the tissue is felt to be more friable than a healthy lung.

Microscopically.– The capillaries in the alveolar walls are very prominent, being distended with blood. The mucous lining of the bronchioles is also hyperæmic. Within the air cells, numbers of red blood corpuscles are observed floating in the serous exudation; these have escaped from the capillaries, many of which have ruptured. This sanguinolent effusion imparts the rusty colour to the sputum, which is so characteristic of an incipient attack of pneumonia. This stage commonly lasts for 36 or 48 hours, and then merges into the second stage.

(2) RED HEPATIZATION.

To the **naked eye** the lung is still expanded and red in colour, but the redness is mottled with fine areas of a lighter colour, which gives the surface an aspect resembling red granite. The organ now feels firm and consolidated, and possesses very much the consistence of the liver; it is on account of this resemblance that the term "red hepatization" is applied. The tissue does not crepitate under pressure, being quite solid; and accordingly pieces of it sink in water. On scraping the surface of a section with a knife, small solid particles are projected on to the blade, and these prove to be casts of the air cells and infundibula. The pleura over the hepatized lung is usually in a condition of inflammation, being covered with a fine layer of coagulable lymph.

Microscopically.—The air cells are distended with a fibrinous exudation, which has coagulated to form a delicate network of fibrin. The filaments of the network ramify in all directions through the air cell, and at the periphery are attached to its walls. Entangled in the meshwork, are various cell elements; these under a high power of the microscope prove to be (1) red blood corpuscles; (2) white blood corpuscles or leucocytes; and (3) epithelial plates which have become detached from the wall of the alveolus. The exuded lymph rarely reaches further than the minutest bronchioles, and does not cause casts in the bronchi. Around the capillary blood-vessels in the alveolar wall, there is an exudation of leucocytes; and a slight degree of connective tissue proliferation may be observed, indicative of commencing interstitial change. The congestion of the capillaries has somewhat subsided. The duration of this stage varies, but is considerably longer than the first.

(3) GREY HEPATIZATION.

This develops from the preceding stage by a natural process of gradation. The general characters of the lung are much the same as before, the most obvious difference being of colour. The lower lobe now has a grey mottled appearance, which might be compared to grey granite: the engorgement has subsided. The tissue is even more solid than formerly, and very friable. In the upper lobe, there is very often to be seen red hepatization or congestion. On squeezing the cut surface of the grey portion a small quantity of dirty greyish-yellow fluid exudes, containing fibrinous casts of the infundibula and bronchi. Over the lower lobe the pleura is thickened by inflammation which has extended from the lung.

Microscopically.—The fibrinous exudation is no longer seen as a meshwork; it has broken down and become granular. There is now in the alveoli a preponderance of white blood corpuscles, which by and by obscure the red ones. Accordingly, in some of the alveoli, we observe a granular-looking fibrinous mass lying in the centre of the air cell and quite detached from the wall, being possibly in the course of absorption. Within the mass, large numbers of white corpuscles are observed. In other alveoli, evidently implicated in a more advanced stage, the contents are merely an aggregation of leucocytes, in which catarrhal cells can be distinguished here and there. All congestion has now subsided. Frequently, the epithelial lining of the air cell shows proliferative changes, the apparent origin of the catarrhal cells. The contents of the alveoli at a later period show evidence of commencing degeneration, partly mucoid and partly fatty; this can be demonstrated by the aid of suitable reagents.

(4) RESOLUTION.

When a pneumonic lung has entered upon the stage of resolution, the tissue still appears pale, but is softer and more flabby to touch. On section, a considerable quantity of muco-purulent fluid can be pressed out of the air-cells and passages, and, in the fluid, fatty casts of the bronchioles and alveoli can be distinguished.

Results of Croupous Pneumonia

1 Resolution c̄ [illegible] Abscess
2 Abscess in Lung - Rare
3 Gangrene
4 Interstitial pneumonia
5 Atrophy
6 Phthisis

Microscopically.—The contents of the air cells have become softened and fatty. Part of this material comes to be absorbed and a portion is expectorated. In some cases, the contents become purulent, and they are got rid of in the same way. But it is a long time before the organ returns to normal. When the infundibula with their alveoli are beginning to recover their tone, there is often a reactionary hyperæmia, for the tissue has been weakened by the inflammation, and is consequently less able to resist the blood pressure than formerly.

RESULTS.

(1) Resolution is the common termination of pneumonia, but the lung is usually bound down at parts by pleuritic adhesions. (2) The purulent infiltration may involve the lung tissue, resulting in inflammatory softening and suppuration. This is not common. The suppuration is generally diffuse, although occasionally an actual abscess forms. Abscess of the lung following pneumonia is nearly always fatal, sometimes by hæmorrhage from erosion of a blood-vessel in the abscess wall. In a few instances an abscess has opened into a bronchus, and its contents have been evacuated by expectoration. (3) Gangrene of the lung sometimes forms the issue of a pneumonia, proving rapidly fatal from blood poisoning. It only supervenes in cases of individuals who had previously been debilitated, as for instance the subjects of diabetes and alcoholism. (4) A somewhat frequent result of pneumonia is an interstitial inflammation, involving the deep layer of the pleura, interlobular septa and peribronchial connective tissue. (5) Sometimes the damaged portion of the lung atrophies, and so one meets with atrophy of the lower lobe occasionally. (6) In a number of cases pneumonia has been succeeded by phthisis, along with caseation. But the one is not always to be regarded as a result of the other: such cases are usually mere coincidences, and without doubt the individuals concerned had inherited a predisposition to phthisis.

II. CATARRHAL PNEUMONIA.

SYNONYMS: BRONCHO-PNEUMONIA, LOBULAR PNEUMONIA.

Since catarrhal pneumonia is intimately associated with bronchitis (the form known as "capillary bronchitis") and indeed is very commonly a sequel of that condition, the description of the disease will be deferred until bronchitis has been considered.

III. INTERSTITIAL PNEUMONIAS.

This is a group of pneumonias, mostly of a chronic nature, and in which we can trace the origin to some special irritant. Such irritants may reach the lung by various channels—by the air passages, the blood or lymph stream. Thus, an interstitial pneumonia results from (1) inhalation of divers foreign matters, chiefly in the form of dust; (2)

many animal and vegetable poisons, especially the virus of syphilis, tuberculosis, and glanders, which is conveyed to the lungs by the blood or lymph ; (3) previous inflammation of the lung or pleura, and chronic venous congestion of the lung ; (4) alcoholism.

Characters Common to all Forms.

I. The changes take place in the region of the connective tissue element, which proliferates to a greater or less extent. The growth of connective tissue in some cases takes place in special relation to the deep layer of the pleura and interlobular septa, in other cases round the bronchi and arteries. (The special character of the hypertrophied tissue varies with the nature of the irritant ; thus in some instances it is quite cellular, in others very fibrous ; while in certain forms it is highly vascular, and in others devoid of blood-vessels).

II. The air-containing cells and passages in the lung suffer from the invasive growth of connective tissue around them. The alveoli are compressed and diminished in size, their walls are thickened, and their lining epithelium frequently shows a reversion to the cubical form—a lower type of cell.

III. The bronchi usually persist as a thickened ring of fibrous tissue, and exhibit a caseous like mass lying free in the lumen. Very often, the bronchi become obliterated by a process of fibrous formation very like obliterative endarteritis in a blood-vessel.

IV. The blood vessels are obliterated to a large extent by endarteritis. The results of this are seen in an atrophy of the portions of lung supplied by these blood-vessels, and possibly in fatty degeneration and caseation of the overgrown connective tissue.

A. PNEUMONO-KONIOSES or DUST PNEUMONIAS.

These terms signify interstitial pneumonias which have resulted from the inhalation of any kind of dust.

I. ANTHRACOSIS or COAL MINERS' LUNG

Is the commonest form met with in this country, and is caused by the irritant action of particles of coal or soot on the lung texture.

Characters.—The lung is much enlarged and increased in weight, and is more solid than normal. The pleura is usually adherent to the chest wall at several points. That membrane seems at first sight to be uniformly black, but the black appearance is found to be due to an immense aggregation of small black nodules, from which lines radiate in various directions, corresponding with the lymphatics of the interlobular septa. On section, the lung is seen to be densely studded with similar black nodules, these being in each case accumulations of carbon

pigment. The tissue is firm and tough, and has a harsh feeling due to the presence of areas of emphysema around the pigment nodules. The organ is œdematous; the fluid which exudes from a fresh section is inky black, and imparts a black stain to the hands. The bronchial glands are enlarged and deeply pigmented with the same black particles. The bronchi often show chronic catarrh, but the mucous membrane does not become black. Sometimes excavations are found, having evidently been produced by an extension of ulceration from the bronchi, with which they communicate.

Microscopically.—In the pleura the pigment is seen to be limited to the deep layer, there being very few nodules in the superficial layer. The nodules here, as well as in the lung substance, are made up of a mass of carbon particles; when highly magnified, many of these particles prove to be granules of coal. From the pleura we can trace the pigmentation along the interlobular septa, within the lymphatic spaces, and thence to the perivascular and peribronchial tissue. Wherever the pigment exists it is surrounded by fibrous thickening, and the interlobular septa come to form great fibrous bands which broadly map out the lobules, and at the same time diminish their space. The inter-alveolar septa are considerably thickened, and contain deposit of pigment. The proliferated and detached epithelial cells are observed lying free in the alveolus, and many of the cells are laden with the black pigment. This evidently points to the mode in which pigment is deposited in the lung, for the catarrhal cells no doubt absorb the pigment (after the manner of amœboid cells) and convey it into the lymphatics, first of all into the perivascular and peribronchial lymphatics, then into those of the interlobular septa, and latterly into the lymphatics of the pleura. Throughout these various situations the lymph channels form a continuous system. The fibrous thickening which is occasioned in the perivascular lymphatics results in the arteries being surrounded by dense fibrous nodules. Within the arteries a process of endarteritis is set up; layer upon layer of proliferated flattened cells is developed until the lumen of the vessel is very much narrowed, in some cases obliterated. In this way the vessels undergo a complete metamorphosis into mere fibrous nodules, which may afterwards degenerate and break down in the centre, leaving a small cavity bounded by ragged fibrous blackened walls. Around the bronchi the fibrous thickening is not so extreme; the bronchial mucous membrane shows evidence of bronchitis.

In very advanced cases of anthracosis, the lung tissue is completely replaced by pigment and fibrous tissue. But in all degrees of the condition there is an absence of congestion with very little cellular proliferation; indeed the process resembles very closely that which occurs in old age from the accumulation of pigment.

II. SILICOSIS OR CHALICOSIS (STONEMASON'S LUNG).

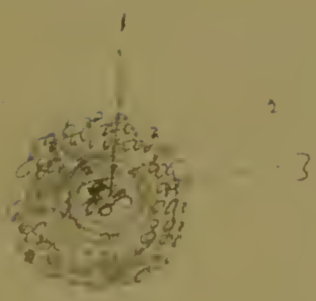
This occurs in persons who work among stone, particularly in masons and quarrymen.

Characters.—These bear a general resemblance to the morbid processes in anthracosis, differing from the latter mainly in degree. Particles of stone, from their shape, hardness, and angular outline, are more irritating than carbon particles, and in consequence produce much more rapid changes in the lung, the resulting fibrous overgrowth being largely cellular and possessing considerable vascularity.

As in the former case, the pleura is thickened and adherent at a number of points, and these adhesions are commonly so tough that the costal pleura remains tenaciously in contact with the thoracic wall when the lung is removed from the chest. The organ is heavy and very firm. Dotted over its surface are numbers of prominent fibrous nodules, many of them ⅓ of an inch in diameter, and therefore much larger than the nodules found on a coal miner's lung. On section, large numbers of similar nodules are found: they are of a grey or bluish colour, with a yellow or light coloured glistening centre, and are bounded by a zone of pigmented lung tissue. When a nodule is cut across, it feels tough and gritty. Between the nodules, the lung tissue is congested and emphysematous. The bronchi are in a state of inflammation, and at some parts excavations are observed which prove to be bronchiectatic cavities (*vide* "Bronchiectasis"). These cavities are pyramidal in shape, the apex of the pyramid opening into a bronchus, while the base is directed to the periphery of the organ: their walls are composed of dense fibrous tissue, from which the thickened interlobular septa radiate in various directions.

Microscopically.—The newly formed connective tissue, wherever found, is of a highly cellular character, being composed of large, mostly elongated cells containing one or more nuclei and particles of stone in their interior. A typical nodule is composed of (1) an aggregation of stony particles enclosed in (2) a number of layers of concentrically arranged fibrous tissue, partly cellular and firmly compacted; the whole surrounded by (3) a zone of exceedingly vascular and cellular tissue. The air-cells show marked catarrh, and are somewhat diminished in size from the thickening of the interalveolar septa, which latter are congested in common with other parts. This congestion appears to be in some measure due to the obliteration of many of the branches of the pulmonary vein. The lymphatics throughout the lung are distended and in part obliterated.

Clinically, a case of silicosis closely resembles phthisis, and it is not uncommon for the former to terminate in the latter condition. The lung structure under such circumstances exhibits typical giant cell systems in the early stages.



Various other forms of interstitial pneumonia caused by the inhalation of foreign particles are met with in **needle-grinders, file-cutters, flax-dressers,** and other artisans. The morbid changes induced in the lungs resemble more or less those above mentioned.

B. INTERSTITIAL PNEUMONIAS from ANIMAL POISIONS.

GLANDERS.

A general description of this disease has already been given in connection with "Vegetable Parasites" (*v.* page 71). There it was stated that the disease usually runs an acute course in man, involving the lungs secondarily, and resulting in the formation of multiple abscesses in their interior. The pulmonary lesion along with the other phenomena bear a striking resemblance to the effects of pyæmia. In horses the disease is oftener of a chronic nature, and in the lungs an interstitial growth takes place along the peribronchial and perivascular lymphatics chiefly.

TUBERCULOSIS.

(See page 175).

SYPHILIS.

Syphilis is not a common disease of the lungs, and when it does occur is usually of the congenital form. So it is met with more frequently in children than adults. The effects of the disease are manifested in different ways, of which three forms may be specified.

I. **Diffuse Interstitial Pneumonia.**—This is generally seen in infants who have died of congenital syphilis, and as a rule affects one lung only. The organ becomes consolidated as in other pneumonias: when cut across it presents an aspect very similar to the appearance of grey hepatization in lobar pneumonia, but, being whiter, has received the descriptive name of "white hepatization." But the latter is further distinguished from the other by being very tough, due to the existence of fibrous bands intersecting the substance in various directions. Microscopically, there is seen to be a wide distribution of new connective tissue, which is very cellular and vascular. The branches of the pulmonary artery are obliterated, but there is no bronchitis—a point of distinction between this and other pneumonias.

II. **Gummata** occur but rarely in the lungs, and are nearly always congenital. They lead to the formation of cicatrices. Occasionally a gumma caseates and breaks down in the centre to form a cavity.

III. **Localized Interstitial Pneumonia** is sometimes set up by the syphilitic virus. It results in the same way as a gumma, causing cicatricial depressions and corresponding deformity of the lung.

C. INTERSTITIAL PNEUMONIAS SECONDARY to ACUTE PNEUMONIA or PLEURISY.

In rare cases of lobar pneumonia, an interstitial process supervenes, producing induration of the affected parts of the lung. There is a fibrous overgrowth which chiefly affects the alveolar walls, invading the air cells and obliterating them by a species of organization similar to that observed in a thrombosed vein.

Acute broncho-pneumonia in children is followed by an interstitial inflammation very like that in syphilitic pneumonia, though not so intense. Some degree of collapse usually occurs along with it.

"Pleurogenous pneumonia" is a form of pneumonia which follows a pleurisy secondary to some mediastinal irritation. It is really either of syphilitic or tubercular origin.

"CIRRHOSIS OF THE LUNG."

This is a name applied to a chronic interstitial pneumonia of uncertain origin. It may be the sequel of an acute pneumonia; but there are many cases where there is no such antecedent, and of which the origin is doubtful.

Characters.—As a rule, one lung only is affected, but both may be involved, and when that is the case one is considerably further advanced in its morbid changes than the other. On examining a cirrhotic lung, it is found to be of small size, and is enveloped in an enormously thickened pleura. The organ feels almost like a piece of indiarubber, being tough and fibrous. On section, there is seen to be a very dense fibrous growth along the lines of the interlobular septa, and around the bronchi and blood-vessels, causing contraction and crowding together of the various structures. The blood-vessels show dilatation, and the bronchi are dilated into bronchiectatic cavities. The **microscopic** appearances are very similar to those met with in stone mason's lung.

III. DISEASES of the AIR PASSAGES.

A. THE LARYNX.

LARYNGITIS,

Or inflammation of the larynx, presents a variety of forms, of which three may be mentioned

I. **Catarrh** of the larynx (as well as of the trachea) occurs commonly as a result of "catching cold," and presents all degrees of severity. It may also result from the inhalation of irritating gases; and it takes place in the course of many specific and infectious diseases, such as scarlet fever, typhoid fever, measles, and smallpox.

Characters.—The inflammation may be acute or chronic, and leads to more or less redness and swelling of the mucous membrane, particu-

larly in the region of the ventricular bands. As a rule the swelling is not much, and so no serious obstruction to breathing is caused; but in children it may be the means of producing suffocation, acting partly mechanically, and partly by inducing spasm of the muscles of the larynx. A mucous discharge is thrown off at first, and this tends to become purulent. The microscopic appearances are those of ordinary catarrh (*v.* page 29).

II. **Œdema Glottidis.**—This name is applied to an œdematous swelling involving the larynx, and often arising with great suddenness. It is usually of an inflammatory nature, though not invariably so. A *simple* œdema sometimes supervenes on Bright's disease and on disease of the heart. *Inflammatory* œdema originates in many ways. Thus, we may enumerate the following modes of origin :—(1) Physico-chemical irritants, *e.g.*, boiling water and corrosive poisons ; (2) Specific irritants, *e.g.*, of diphtheria or smallpox ; (3) Disease of the laryngeal cartilages ; (4) Extension of inflammation from the fauces or pharynx. Suppuration in the tonsil sometimes causes œdema glottidis, and it frequently supervenes after operations on the throat ; (5) Complication of tumour growth in the region of the larynx ; (6) Spontaneous, of undiscoverable origin. Probably many of these cases are really specific, resembling erysipelas.

Œdema glottidis manifests itself specially in those parts of the larynx where sub-mucous tissue exists, vizt., towards the base and cushion of the epiglottis, the aryteno-epiglottic folds, and the false vocal cords. The inflammation is not confined to the mucous membrane, but extends into the sub-mucous tissue in these particular regions. The effect of the swelling is to narrow, often to completely obstruct, the upper opening of the larynx ; the latter contingency, of course, issues in asphyxia. After death the swollen parts appear pale (not hyperæmic), and on incising the swelling a muco-purulent or purulent fluid exudes.

III. **Membranous inflammation.**—This condition is to be regarded as diphtheritic in origin. Diphtheria is a disease which was previously mentioned in connection with the subject of bacteria, .and is generally regarded as being due to the agency of these organisms. It usually starts in the fauces and extends to the larynx, often to the trachea, and even to more distal portions of the respiratory tract. The characteristic feature of the disease is the production of an inflammatory exudation or "false membrane" on the mucous surface, hence the term "membranous" inflammation. The children's disease of **croup** (properly so called) is simply a diphtheria of the larynx, although previously considered and still regarded by many as a separate disease. For convenience, the description of the anatomical changes will be deferred until we come to speak of diphtheria in connection with the fauces.

ULCERATIONS.

Simple ulceration not unfrequently occurs in the larynx after repeated attacks of catarrh. In these chronic cases, flat superficial ulcers are observed, situated for the most part posteriorly. Occasionally, the racemose glands ulcerate, and they can be seen on the epiglottis.

Tubercular ulceration of the larynx is a very important condition, so much so that in cases of extreme degree it is spoken of as "laryngeal phthisis." It is always associated with pulmonary phthisis, usually in a secondary manner, and it is found as a matter of experience that in one out of every three cases of pulmonary phthisis the larynx is involved. Sometimes the lesion in the larynx is more pronounced than the pulmonary lesion, and in particular instances the larynx is apparently affected primarily.

The tubercular disease attacks chiefly the epiglottis, the aryteno-epiglottic folds, and the ventricular bands. In these situations the mucous membrane first becomes pale and then swollen. The thickening is of an œdematous nature, and microscopic examination reveals tubercle granulations scattered through the inflammatory exudation. In course of time minute ulcers form in relation to the superficial tubercles, which caseate and soften. These ulcerated points coalesce to form larger ulcers, which to begin with are merely superficial, but latterly cause considerable destruction of tissue. Between and around the ulcers there is thickened mucous membrane. The vocal cords frequently become involved, ulcers form on them, and they have even been destroyed, resulting in a loss of voice. The cartilages are sometimes implicated, and suppuration may ensue. As a rule, the trachea exhibits similar ulceration.

Syphilitic ulceration.—Syphilis induces inflammatory changes in the larynx, which are met with in all degrees of severity. In many cases there is only a passing catarrh, but in the later manifestations of the disease the mucous membrane becomes thickened and then ulcerated. This affects the epiglottis especially, but the process tends to spread to other parts of the larynx. The swelling may of itself lead to obstruction of the larynx, necessitating the performance of the operation of tracheotomy. In the granulation tissue, there is a constant production of connective tissue, which, favoured by a healing tendency, contracts and causes deformity of the larynx—so great sometimes as to produce complete stenosis.

TUMOURS.

Of **innocent** tumours, the one most commonly found in the larynx is **papilloma**. They often grow apparently as the result of a simple inflammation and "are particularly common in persons who, from the nature of their profession, use the voice frequently." They usually grow from the vocal cords. In character, they may be either hard and warty, or soft, and are often pedunculated. When excised, they may recur in

malignant form, developing into a sarcoma or cancer. A small **fibroma** is sometimes met with, usually in the form of a firm pedunculated tumour attached to the vocal cord or base of the epiglottis.

Malignant tumours are of occasional occurrence. **Sarcomas** have been observed, usually of the spindle celled variety. **Cancer** occurs both primarily and secondarily. It takes the form of squamous epithelioma, arising usually from the ventricular bands, or by secondary extension from the tongue. These tumours very commonly acquire a cauliflower character, and undergo ulceration centrally, while the peripheral part spreads ; this results in destruction of tissue and consequent deformity.

B. THE TRACHEA AND LARGER BRONCHI.

The morbid conditions which affect the trachea are almost identical with those involving the larger bronchi, and the diseases of each closely resemble those involving the larynx. This fact will be understood when it is remembered that these different portions of the respiratory tract are structurally homologous.

BRONCHITIS.

This signifies catarrh of the larger and middle-sized bronchi. These tubes are subject to be inflamed *per se*, the process not extending into the finer bronchi. That obtains in the case of adults and all who are past the age of childhood ; in children the small capillary bronchi are specially affected. In slight cases the larger bronchi are involved, usually along with the larynx and trachea, but in typical bronchitis we find the middle-sized tubes in particular to be the seat of catarrhal changes. The inflammation may be *acute* or *chronic*.

The conditions under which bronchitis occurs are not always evident. In many instances, no more specific cause can be elicited than "a cold," and in the case of many individuals exposure to cold is very liable to bring on a recurrence of the condition. In persons afflicted with an evident predisposition to bronchitis, very trivial ailments, such as disorders of the stomach, are sufficient to set up the inflammation anew. Bronchitis is very frequently met with in cases of measles, whooping cough, etc. It was pointed out also that one of the most frequent results of mitral disease is a persistent bronchial catarrh, which becomes chronic.

Characters.—These vary with the chronicity of the condition. An acute catarrh causes first of all redness and swelling of the mucous membrane. There is an exudation thrown out on the mucous surface ; this at first consists merely of concentrated mucus, tough, scanty, and transparent. By degrees the exudation (which is expectorated as "sputum") becomes more abundant, and at the same time less tenacious and transparent. When the disease is at its height, the sputum is of a yellowish white colour, resembling pus, but being more tenacious; it is then spoken of as "sputum coctum." As the inflammation becomes chronic, the exudation tends to be more abundant, and partakes of a muco-serous character, being for the most part composed of serum; it has therefore lost its tenacity, as well as much of its opacity, and is in addition very commonly frothy. Sometimes the sputum becomes purulent. The mucous membrane in chronic bronchitis is of a slaty purple colour, and the secreting glands are sometimes evident in the form of small translucent projections on the surface. The bronchial tubes may exhibit a slightly sacculated character, due to an irregular dilatation.

Microscopically.—In acute cases, we first of all observe a hyperæmia in the vessels beneath the basement membrane, which is pushed inwards. Within twenty-four hours, that membrane becomes swollen and œdematous; along with, or immediately following this, the epithelium proliferates, and its superficial layers desquamate, so that there is no longer a surface exhibiting rows of ciliated columnar cells, but an irregular surface covered with epithelial cells of various shapes and sizes. The mucous glands, which are in a state of hyper-secretion, are seen to contain an excessive number of chalice cells. When the catarrh is at its height, one can see the greatly swollen basement membrane, upon it a flattened layer of cells (Debove's layer) surmounted by a number of irregularly disposed layers of proliferated epithelial cells. Around the distended vessels in the inner fibrous coat are numbers of leucocytes, mixed with connective tissue corpuscles. In chronic cases, the external fibrous coat shows great cellular infiltration, and in the muscular coat there is a similar infiltration which becomes the means of weakening the muscular coat, and so allowing the bronchus to dilate. The muscular coat as a result appears thin and atrophied. The exudation at first consists of mucus, which soon gets loaded with catarrhal cells. These cells gradually increase in number, while the fluid becomes more serous. The degree of toughness depends on the proportion of of mucin in the exudation, and the opacity on the number of the cells. "Sputum coctum" is a muco-serous fluid containing great numbers of round cells densely massed together, among which are a smaller number of cells evidently epithelial in character. The nature of the round cells is disputed; some hold them to be leucocytes solely, but no doubt many of them are epithelial cells derived from proliferation of the mucous membrane of the bronchus itself and of its mucous glands. The glands very often degenerate as a result of the persistent catarrh.

Chronic bronchitis leads to dilatation of the bronchi, and very often to emphysema of the lung (*v.* page 152). Sometimes a peri-bronchitis is set up, resulting in fibrous thickening of the bronchial wall, and this may be followed by further changes in the corresponding parts of the lung.

Cause.—Children are very liable to be attacked with capillary bronchitis after measles or whooping cough, less commonly after smallpox or diphtheria. Sometimes it arises secondarily by direct extension from the larger bronchi, but this is comparatively rare. In adults, the disease may be a sequel of typhoid fever or erysipelas, or it may result from the inhalation of foreign matter in the shape of irritating gases, blood, or minute solid particles. It also occurs as a factor in "hypostatic pneumonia."

Characters.—The condition commences with a catarrh of the finer intralobular bronchi, and this is succeeded by a peribronchitis, then by a catarrhal pneumonia, constituting what is known as "broncho-pneumonia." In fact, capillary bronchitis is nothing more nor less than the initiatory stage of broncho-pneumonia, and so we shall proceed to a consideration of the latter.

BRONCHO-PNEUMONIA.

(SYNONYMS: CATARRHAL—LOBULAR PNEUMONIA).

This condition being a direct sequel of capillary bronchitis, it is unnecessary to say anything further as to its causation. But it is essential to understand how the pneumonia supervenes on the bronchitis. In some cases the bronchitic exudation is carried into the infundibula and their alveoli by the force of inspiration, and there induces inflammatory changes by direct irritation. In other cases, the inflammation apparently spreads through the bronchial wall. In a large number of cases the bronchitis leads to collapse, and a pneumonic process arises in the collapsed pulmonary tissue. Collapse of the lung is one of the most frequent antecedents of broncho-pneumonia.

Morbid Phenomena.—The disease invariably assumes a lobular form, on account of the mode of propagation from bronchi to lung tissue. The capillary bronchitis which ushers in the condition is in a general way comparable to an ordinary bronchitis, but with certain differences. The congestion in and around the bronchial walls is much more extreme: the walls are greatly thickened in consequence, and hæmorrhage is prone to occur from the distended capillaries. The proliferation and desquamation of epithelium is sometimes so great as to obstruct the lumen of the tube. The wall of the bronchiole becomes densely infiltrated with leucocytes, which extend into the adjacent alveoli. Then the pneumonic process supervenes, for the effect of the bronchitis is to produce secondary inflammatory changes in those parts of the lung which correspond with the affected bronchioles. The pneumonia may or may not be preceded by collapse. Whatever the mode of extension may be, the result is a catarrhal pneumonia usually involving a lobule or lobules. The alveoli are greatly congested, the hyperæmia being especially marked in those air cells in closest proximity to the bronchioles. Small extravasations

commonly occur into the alveoli. They gradually become filled with catarrhal products, large numbers of leucocytes, and sometimes fibrinous material, leading to condensation of the lung. These condensed parts frequently have a wedge-shaped configuration, corresponding to a lobule, and are apt to be mistaken for areas of collapse. Between the consolidated patches the lung tissue is much congested, the interlobular septa especially being swollen and œdematous looking. Very frequently the interlobular septa as well as the peribronchial connective tissue become the seat of rapid inflammatory changes, resulting in an acute interstitial pneumonia.

The **naked eye** appearance of the lung varies. In a typical case, one finds areas of collapse scattered through the organ, and involving one or more lobules. These when situated superficially present the usual depressed character. Among them are observed a number of other patches, resembling the former in size but distinguished from them by being raised above the surface, and more granulated in appearance. These are the consolidated pneumonic patches; in an early stage they are deeply congested and highly coloured, but as the process goes on they become paler in colour, simultaneously with the filling up of the air cells with catarrhal products.

Sequelæ.—(1) When broncho-pneumonia occurs during acute diseases, complete resolution and absorption very frequently occurs, *i.e.*, the catarrhal cells degenerate and are absorbed. (2) Sometimes interstitial changes are set up as already pointed out. (3) In cases of hypostatic pneumonia especially, the condition goes on to suppuration, with the formation of small abscesses. This is particularly the case when the broncho-pneumonia has been set up by the retention of secretion. (4) Very rarely the inflammatory products caseate, and only when the disease is complicated with tuberculosis. (5) Occasionally a bronchus ulcerates, dilates to form a bronchiectatic cavity with ragged walls, and containing purulent material.

HYPOSTATIC PNEUMONIA

Is a form of pneumonia which occurs in persons dying from exhausting diseases—such as fevers—from disease of the brain, or from the inhalation of poisonous irritants. The site of the inflammation is governed by the recumbent position of the patient. "It tends to involve larger tracts of the lung, and is a mixed condition of broncho-pneumonia, congestion, and croupous exudation."

TUBERCULOSIS OF THE LUNG.

This is a disease now well known to be produced by the *bacillus tuberculosis*. That organism gains access to the lungs by one of three channels, vizt. :—(1) the air passages; (2) the blood-vessels; or (3) the lymphatics. In any case it sets up a tubercular process, which may be acute or chronic.

A. ACUTE TUBERCULOSIS.

We mean by this, cases where death occurs in a short time from pulmonary symptoms, or from tubercular meningitis, occurring secondarily to tubercular disease in other organs.

The **morbid phenomena** vary with the mode of introduction of the virus, and also with its mode of extension; but in all forms, we meet with tubercle granulations disposed in one form or another in the lung tissue. Sometimes they are widely distributed over the whole organ, at other times they are grouped and localized at particular parts.

I. Infection and Extension by Lymphatics.

The lymphatic glands are very often the first structures in the body to be affected with the tubercular virus. In children this very constantly happens in the case of the mesenteric glands, from which the infection spreads upwards through the diaphragm to the lymphatic vessels in the deep layer of the pleura. The cervical glands are also a common seat of early infection. The bronchial glands often receive the infection first and communicate it to the lymphatic system of the lung.

In such cases, there is an extensive development of grey tubercle granulations along the course of the lymphatic vessels, and the granulations are especially evident in the deep layer of the pleura, the interlobular septa, and the peribronchial tissue. They resemble pepper grains, and a section of the lung appears to be sprinkled with pepper. The lung tissue around the granulations is congested, but the granulations themselves are extra-vascular. The latter possess the ordinary structure of tubercle granulations (*v.* page 68). Frequently they induce catarrhal changes in the adjacent air cells and an inflammatory infiltration around.

Sometimes, the tubercle invades the peribronchial lymphatics chiefly, giving rise to the condition of **tubercular peri-bronchitis.** That occurs in relation to the medium-sized bronchi. This always tends to become chronic, and consequently one finds distinct giant cell systems at various points. But for this latter circumstance, the anatomical changes are very similar to an ordinary chronic fibrous peri-bronchitis; the wall of the bronchus is greatly thickened, and its lumen is invaded, with the frequent result that the tube becomes obstructed. The obstructing mass tends to become caseous. The result of this obstruction may be collapse, but very frequently the tubercular material is inhaled into the alveoli and sets up a tubercular broncho-pneumonia. (This is one of the commonest lesions in chronic phthisis).

II. Infection and Extension by Blood-Vessels.

Here also there is a growth of tubercle granulations, but (as might be anticipated) they are very widely diffused throughout the lung. Otherwise the morbid phenomena are identical with the former. The

tubercles are most abundant towards the root and apices of the lung. With the aid of the microscope, the tubercular granulation can often be recognized to take origin in the epithelium of the capillaries, or of the air cells.

III. INFECTION AND EXTENSION BY THE AIR PASSAGES.

In many cases the tubercular virus is inhaled. It may be drawn right into the alveoli, and thence gain access to the lymphatics; or it may attack the bronchial wall. Secondary infection may occur by the inhalation of sputum from one bronchus to another. The usual result of such infection is primarily a capillary bronchitis, which leads to **tubercular broncho-pneumonia.** This is the commonest lesion in the lung in acute tuberculosis in children, as well as in rapid phthisis pulmonalis in the adult. Let us compare it with ordinary broncho-pneumonia. In the latter condition there is primarily a catarrh of the capillary bronchi with cellular infiltration of the bronchial wall, extending to the interlobular septa. These phenomena are also observed in the tubercular form, but there is a more extensive cellular infiltration both of the bronchial and alveolar wall, producing thickening and induration, and latterly softening and caseation. In that way, the structure of these parts becomes obscured, and in course of time the air cells which surround the bronchiole may become completely metamorphosed into a solid caseous mass. The cells at the more peripheral part of the lobule exhibit catarrhal changes, often mixed with fibrinous exudation. Tubercle bacilli are found in large numbers in the caseated area, less abundantly in the surrounding parts further from the bronchiole. To the naked eye, a section of the lung exhibits great numbers of granulations varying in size from $\frac{1}{16}$ to $\frac{1}{4}$ of an inch in diameter, greyish white in colour, with a yellow softened centre. They are often grouped in clusters, corresponding to the branches of the bronchi.

B. CHRONIC TUBERCULOSIS.

In slowly progressing cases of tuberculosis, the lung is invaded by typical giant cell tubercles. These spread mainly by way of the lymphatics, and invade the air containing tissue. The condition is not so wide-spread as in the acute process, being localized in great degree, and involving especially the root and apex of the lung.

TUBERCULOSIS OF BRONCHIAL GLANDS.

The bronchial glands are practically always affected in tuberculosis of the lung. They usually become enlarged. If the condition be acute, they tend to soften and show caseating suppuration. In chronic cases, the caseous material is often impregnated with lime salts, imparting a mortar-like consistence to the gland, sometimes causing it to become quite calcified. In other instances, the glands become the seat of pigmented nodules of fibrous tubercle.

PHTHISIS PULMONALIS.

This name indicates a group of morbid processes in the lung, among which a destruction of the lung tissue is the most prominent feature. They are mainly due to the agency of the *tubercle bacillus*, and therefore tubercular processes play a very important part amidst the many other phenomena which are manifested in the lungs. There is coincidently a great wasting of the body, and the emaciation very often terminates in death.

Certain preliminary facts should be noted. In the first place, both lungs are usually involved, though one may be in a more advanced stage than the other, and the lesions in each may vary in character. Secondly, the disease in the majority of cases starts towards the apex of the lung, but in many cases where the indication is at the apex there has been an extension from the root of the lung. From the apex, the process extends by direct continuity to the base. Thirdly, the pleura as a rule is involved either by the tubercular affection, or by a simple chronic inflammation leading to the formation of adhesions. Such adhesions may bind the lung to the chest wall at any point, or the lobes of a lung to one another. In the fourth place, the disease is largely an *inflammatory* one; the inflammatory products lead primarily to a **consolidation** of the lung, and the consolidated parts afterwards break down and become excavated to form **cavities**.

I. CONSOLIDATION OF LUNG.

In all forms of phthisis, the consolidation of the lung proceeds by the formation of nodules, which by their subsequent coalescence give rise to large solid areas of pulmonary tissue. The finer bronchi are probably the first to be affected, and from them the process extends either into the alveoli or through the bronchial wall into the surrounding connective tissue. In the former case a catarrhal inflammation of the air cells is quickly set up, while in the latter the tubercles tend to undergo fibrous changes. The nodules vary a good deal in character. Thus, there are (1) small grey nodules growing in the interlobular septa, and consisting mostly of giant cell systems with surrounding infiltration. (2) Firm fibrous nodules of chronic duration, mainly peribronchial in position, much pigmented, often encapsuled by a fibrous zone in which are giant cell systems. (3) Soft caseous granulations, resembling the caseous broncho-pneumonic nodules in acute tuberculosis, and formed in exactly the same way. They are found in acute phthisis. Between them, the pulmonary tissue exhibits a fibrinous and catarrhal exudation. (4) Larger nodules found in chronic cases, at first grey and translucent, afterwards yellow and soft. They often assume a racemose arrangement, like branches of a shrub. These nodules consist of chronic fibroid tubercle, with abundant giant cell systems, and are usually surrounded by chronic interstitial pneumonia. Whatever the particular form of lesion in the

lung, and whether it be acute or chronic, each is related to the other by the fact that tubercle bacilli are present in all forms.

II. DESTRUCTION OF LUNG AND FORMATION OF CAVITIES.

The lung tissue usually begins to degenerate at the apex, and it is there that a cavity first forms. The method of their formation varies in different cases, and there are three recognized modes of origin. (1) The commonest mode is by a softening and absorption of caseated areas of tissue. A number of small cavities may in that way arise at different points, and by their coalescence give origin to a large irregular cavity. During the process of formation, a peripheral growth of granulation tissue is induced, and this comes to form the lining membrane of the cavity. (2) Sometimes the cavities prove to be true bronchiectatic cavities. As before pointed out, these are produced in chronic phthisis by traction of the cicatricial tissue on the bronchial wall. (3) In many cases, cavities originate by a process of softening and ulceration in a bronchus. That obtains in acute phthisis especially.

Phthisical cavities exist in very varying shapes and sizes. They usually communicate with a bronchus, through which they can evacuate their contents. The nature of their walls depends largely on the origin of the cavity, and its rapidity of formation. Frequently a cavity is in the form of a ragged ill-defined space in the midst of caseating tissue: that is so in acute phthisis. On the other hand they may have well-defined fibrous walls, and such is the case in chronic phthisis. In these the fibrous wall is lined by a smooth, glistening membrane which (in the recent lung) is covered by a layer of caseous pus. Further, within the walls, and often traversing their interior, one commonly sees the remains of interlobular septa, or bronchi, or blood-vessels, and which appear in the form of fibrous bands. In some cases we find a wall composed of soft œdematous connective tissue, almost like myxomatous tissue.

The **sputum** of phthisis varies at different stages of the disease. In the earlier stages, it is composed of mucus with catarrhal cells and leucocytes, and is frequently tinged with blood. The blood is derived from the mucous membrane of the bronchioles mainly, and is caused by the inflammatory hyperæmia, possibly aided by ulceration. After cavities have formed, the sputum is largely made up of purulent matter, in which fragments of elastic tissue and other elements of the lung can be discovered. Bacilli can usually be detected by suitable staining.

Sequelæ.—It is possible to cure phthisis. The progress of the disease may be arrested, and moreover parts which are already caseous may dry up and become encapsuled by fibrous tissue. Following upon this the caseated tissue may become calcified. Instances of this are frequently met with, where in the apex of an otherwise normal lung a cicatrix is discovered, marking the site of a calcified mass embedded in the lung tissue.

A cavity often extends so widely as to perforate the pleura. The result of this is that the purulent contents are discharged into the pleura, air is admitted simultaneously, and a pyo-pneumothorax is set up. In many instances the cavity is prevented from rupturing by the inflammatory reaction which it occasions in the pleura, whereby the membrane undergoes a local thickening and patches up the lung as it were.

The bronchus with which a cavity communicates often shows tubercular ulceration of its mucous membrane, caused by frequent contact with the irritating tubercular expectoration. Further, since the patient (especially if a child) is always liable to swallow his sputum, the intestines very frequently become infected.

The blood-vessels in or around a cavity are exposed to the necrotic process, and are often ulcerated through, allowing of free hæmorrhage. In many cases their walls are so weakened that they yield beneath the blood pressure, and an aneurism forms on them. Such aneurisms burst in course of time, and are a frequent source of fatal hæmorrhage.

Forms of Phthisis.—The variations of type observed in different cases of phthisis are chiefly dependent on the chronicity of the process and the degree of attendant inflammation.

I. **Acute rapid phthisis** is manifested in two principal forms, in either of which the entire lung may become metamorphosed into a caseous solid mass. In the one case, there is a tubercular process complicated with croupous pneumonia. The other may be described as a confluent tubercular broncho-pneumonia; it is frequently spoken of as "caseating catarrhal pneumonia."

II. **Subacute phthisis** is a form in which there is a steady progress from apex to base, an extension by means of nodules, and a gradual destruction.

III. **Chronic phthisis.**—Here there is a slow and progressive consolidation of the lung, followed by destruction. There is a variety of it known as "fibroid phthisis," in which we find a large amount of fibrous consolidation.

PARASITES.

Animal parasites are of little importance in the human lung. The only one worthy of mention is the *tænia echinococcus*, which is found only in the cystic form, constituting the hydatid cyst. These hydatids are usually solitary, and are mostly situated in the lower lobe. They are met with more frequently in Australia and Iceland than in other parts.

TUMOURS.

Primary tumours of the lung are rare, and there are only two forms of any consequence that have been observed. **Columnar epithelioma** may arise at any part of the organ, and it usually invades large

ts in the formation of pustule-like patches on the
membrane of the mouth. Sometimes the
[illegible]

tracts of the lung tissue. **Lymphosarcoma** is a tumour which invariably starts in the bronchial glands, or in the adenoid tissue which is disseminated throughout the organ. It then extends and infiltrates the lung.

Secondary.—Many forms of **cancer** and **sarcoma** grow secondarily in the lung, frequently involving the pleura also. Cancers usually develop first in the lymphatic glands, and they are late of appearing in the lung tissue proper—in fact, they are sometimes spoken of as "tertiary" tumours. Occasionally, the cancerous material reaches the lung in the shape of emboli. Cancers in the lung are generally multiple, and they repeat the structure of the primary tumour. Sarcomas of nearly every variety have been found in the lung. They also are multiple as a rule, and exhibit the same structure as the primary growth.

DISEASES OF THE ALIMENTARY SYSTEM.

A. MOUTH and TONGUE.

INFLAMMATION

Of the mouth is met with in more than one form.

Catarrh is set up by various kinds of irritants: (1) mechanical, *e.g.*, a decayed tooth; (2) chemical, *e.g.*, mercury and arsenic; (3) specific, *e.g.*, the virus of scarlet fever, typhus, and smallpox.

The **morbid phenomena** are those of simple catarrh, but the appearances produced vary in different cases. There is always more or less desquamation of epithelium, with mucous and serous exudation: these are mixed in varying proportions. On the tongue there is a preponderance of epithelium, and this often forms a light-coloured layer on the surface, constituting a "furred tongue." In cases of fever, when the patient lies with the mouth open, the catarrhal products become dried and discoloured, forming a coating of "sordes" over the tongue and gums.

In *smallpox*, the catarrh is supplemented by the formation of pustules, similar to those on the skin. In *scurvy*, the gums are especially affected; they become swollen and œdematous, hæmorrhage takes place from the distended capillaries, and ulcers form at the margin of the gums.

Follicular Stomatitis is a name applied to inflammation localized in and around the mucous glands in the mouth. It is specially seen in children when teething, also after measles. The inflammation is characterized by the formation of small vesicles, which generally burst and form ulcers.

Aphthous Stomatitis or "Thrush." This disease has been already mentioned in connection with the parasite *saccharomyces albicans* (*v.* page 76).

Gangrenous Stomatitis, or "Cancrum Oris," has also been briefly mentioned (*v.* page 17). We may add here that the disease occurs in children who have been debilitated by fever and bad nourishment, and that it would appear to be of specific origin. It manifests itself first by an ulceration on the inside of the cheek, attended with a diffuse inflammatory swelling of the side of the face. As the ulceration proceeds within, the cheek on the outside gradually blackens and then sloughs: in this way the cheek is perforated. The process does not stop here, unless the patient dies. The destruction of the face progresses, and the jaw may necrose. The disease is always fatal, often from septic poisoning.

SYPHILIS

Produces various lesions in the mouth. A primary hard chancre is frequently met with on the lip; it forms a tumour resembling an epithelioma. In the second stage, there is usually some degree of catarrh evident. "Flat condylomata are particularly frequent in the mouth, and they often break down and form superficial ulcers, especially on the lower lip and angle of the mouth." Gummata are also common; when they heal they cause cicatricial contraction and deformity.

TUMOURS.

Angiomas sometimes grow on the lips, as well as on the tongue. **Papillomas** occur in various parts of the mouth. Soft tumours grow on the mucous membrane of the lips, while at the junction of skin and mucous membrane hard warts are frequently found. On the tongue papillary growths have been observed in the form of long slender filaments like hairs: these are simply the normal papillæ greatly elongated.

Of malignant tumous, **cancer** is very common. An **epithelioma** of the lip is one of the commonest of all tumours; it occurs almost exclusively in the male sex, and is no doubt often set up by the smoking of a short clay pipe, or by the irritation of shaving. Epitheliomata are also frequently met with on the tongue. They owe their origin to direct irritation in most cases: their usual situation is at the edge of the tongue, and in very many instances the sharp prominent edge of a decayed tooth can be discovered exactly opposite the tumour.

Malignant epulis is a tumour of sarcomatous nature which grows from the aveolar processes of either jaw, and projects into the mouth, often displacing the teeth. Microscopically, these tumours are composed mainly of spindle cells, with a few giant cells interspersed.

Macroglossia, a form of enlargement of the tongue, has been already considered. It is often accompanied by enlargement of the lips.

Ranula has been mentioned in connection with cysts (*v.* page 56).

B. *The FAUCES.*

INFLAMMATION.

As in the mouth, there are various forms of inflammation of the fauces.

Acute catarrh of the fauces constitutes the most ordinary form of that common affection designated by the term "sore throat." It is a very constant result of catching cold, and is an important symptom in many acute febrile diseases. A simple catarrh exhibits no special characteristics, but in diseases like smallpox and diphtheria the catarrh develops into much graver manifestations.

Chronic Catarrh is usually a sequel of repeated attacks of acute inflammation. It leads to different phenomena in different instances. Sometimes it takes the form of "relaxed throat," which is characterized by a soft uniform succulent swelling of the mucous membrane. The uvula is often greatly elongated as a result of the inflammation. In other cases the swollen membrane is studded with prominent granulations, which are connected either with the mucous glands or lymph follicles. These sometimes ulcerate. Catarrhal inflammation of this kind is usually caused by excessive and loud use of the voice, and so is met with in public speakers and singers.

Phlegmonous Inflammation of the fauces is a common occurrence. It involves both the mucous and sub-mucous tissue, and usually begins on one side in the region of the tonsil, from which the inflammation extends to the pharynx, sometimes to the glottis. It leads to intense redness and swelling of the parts, and frequently ends in the formation of an abscess, which may open into the mouth. It may prove fatal by the supervention of œdema glottidis.

The ætiology of the condition is unknown, but from the fact that it sometimes acquires almost an epidemic character, one is justified in assuming the existence of some specific exciting cause, probably bacterial in nature.

Diphtheria.—It was previously stated that this disease commences in the fauces: it is especially manifested in the tonsil and the nasal surface of the soft palate. As to the origin of the condition, there is no doubt that it is produced by the agency of bacteria. These organisms have been found in the mucous membrane of the region involved and in the blood. The disease is infectious, and often terminates fatally.

Characters.—The first sign of inflammation is a catarrh of the mucous membrane. Following this, a number of minute, pale, slightly elevated patches make their appearance on the surface. These soon coalesce to a large extent and form a pellicle or "false membrane," resembling very much a piece of dirty white kid glove. In an early stage of its existence this false membrane can be readily detached by the fingers, leaving a red swollen surface beneath. But the membrane quickly increases in thickness, as well as in extent, and on attempting to detach it sometime later, it is found to be adherent to the surface. If it be forcibly torn away, a bleeding ulcerated surface is exposed: this shows that the membrane does not lie free on the mucous surface, but involves the latter in the process of growth.

From the fauces the membrane extends in various directions. It may grow upwards into the nose, downwards into the œsophagus, more commonly into the larynx and trachea, sometimes reaching to the bronchi and their branches. When the exudation is mainly or entirely limited to the larynx and trachea, the name of **croup** is employed to designate the condition. The membrane which forms in these situations differs 'from that in the fauces, inasmuch as the former never becomes adherent but always remains quite superficial. The same fact holds good with regard to the bronchi, where the exudation is very soft and easily displaced.

Microscopically, an examination of the pharynx in an early stage shows diffuse swelling, particularly of those structures constituting the pharyngeal tonsil. The lymph cells are greatly increased in number. Later, there is evidence of degeneration in the mucous membrane—a commencing necrosis—and masses of micrococci can be discovered in the degenerated tissue. The false membrane at first consists of a number of stratified layers of a homogeneous hyaline material, between which a few round cells are observed: it apparently results from a hyaline swelling of the superficial layers of epithelium. In the advanced stages the mucous membrane is seen to have undergone a complete necrosis, attended with coagulation of the necrosed products. (This is an instance of coagulative necrosis, *v.* page 15). The false membrane is now thickened, and is composed of the degenerated mucous membrane, largely mixed with fibrin, and swarming with micrococci. On its under surface the membrane sends prolongations into the ducts of the glands, hence the reason why it becomes adherent.

The distinctive characteristics between diphtheria and croup are, that in the former the micrococci and the slough are always present, but the marked fibrinous exudation, though usually present is not essential; whilst in croup the exudation is essential and the micrococci if present are found only on the surface, do not invade the membrane or the surrounding lymphatics, and so do not get into the system to give rise to constitutional symptoms. (Woodhead).

Tonsillitis.—An *acute* inflammation of the tonsils occurs independently or as a coincident phenomenon in general inflammations. In the latter case suppuration may follow; in the former the condition tends rather to become chronic. *Chronic* tonsillitis is a very common occurrence in young persons. It results from repeated acute attacks, which

ultimately cause considerable and permanent enlargement of the gland. The enlargement consists mainly in a hyperplasia of the lymph elements in the follicles.

Syphilis produces varying degrees of inflammation in the fauces, from an ordinary catarrh to extensive ulceration and cicatrization. As in the mouth, condylomata are often met with.

C. The PHARYNX and ŒSOPHAGUS.

DIVERTICULA.

A diverticulum means a localized dilatation or pouch. The commonest site for one is on the posterior wall of the pharynx, near its junction with the œsophagus, and the pouch projects posteriorly for a varying distance, depending on its size. How such a pouch comes to exist is a matter of some dubiety, but in all probability it is the result of direct pressure exerted by some abnormal force within the tube. In several instances a diverticulum has been observed to form after the impaction of a large bolus of food in the œsophagus. This theory of their causation is borne out by the fact that diverticula have not been discovered in infants. Further, the posterior wall of the pharynx and œsophagus in the region of their junction is the weakest portion of the entire tube, for at that point the muscular wall is comparatively thin.

A diverticulum is sometimes found on the anterior wall of the œsophagus, behind the bifurcation of the trachea. The origin of such a pouch is quite different from the former. Here it results from inflammatory changes in the bronchial glands : these glands frequently form adhesions with the front of the œsophagus, and then sometimes cicatrize, dragging the anterior œsophageal wall with them as they contract. The result is that a funnel-shaped pouch forms in that situation, and this is liable to subsequent enlargement by the constant passage of food into it.

Microscopically.—A diverticulum does not possess all the coats of the œsophagus. The mucous lining is present, but there are no muscular fibres ; the mucous coat is supported by fibrous tissue merely. Probably in many cases, during the process of formation of the pouch, the mucous coat is pushed through the muscular fibres, and forms a fibrous covering for itself as it advances.

DILATATION AND HYPERTROPHY.

As a result of stricture of the œsophagus in the lower part of its course, the tube undergoes hypertrophy. Stricture may arise in various ways—from tumour growth within, or the pressure of tumours or aneurisms from without. When of prolonged duration, the tube becomes enormously dilated, while its wall simultaneously thickens. The thickening implicates the mucular coat chiefly, to a less extent the mucous membrane; the latter sometimes ulcerates.

The œsophagus has been known to dilate when no stricture existed, and such cases are generally attributed to a chronic inflammation of the tube, which diminishes its resiliency.

INFLAMMATION AND ULCERATION.

The œsophagus being lined with a mucous membrane that is almost devoid of glands, possesses a resisting power to the influence of many irritants; and the irritants must be of a powerful nature to produce any palpable alteration on the interior of the tube.

Catarrh, whether acute or chronic, is of little moment; it may cause some slight thickening of the mucous membrane.

Thrush and **smallpox** each affect the œsophagus in certain cases. The aphthous patches of thrush occasionally spread as far down as the stomach. Smallpox is manifested by the formation of pustules, similar to those which more frequently infest the mouth.

The most important inflammations of the œsophagus are those produced by **physico-chemical irritants,** particularly boiling water, strong acids, and alkalies. Fluids of such a nature set up an intense inflammation in the pharynx and œsophagus, as well as in the stomach. Boiling water causes enormous swelling; acids exert a destructive action, charring the parts and producing variously coloured sloughs; while alkalies, by virtue of their special properties, soften and dissolve the lining of the tube, converting it into a layer of gelatinous slime. The muscular coat is more or less affected, and often destroyed to a large extent. When the patient does not die from the shock of the accident, a very common sequel of the inflammation is cicatrization, and a degree of contraction of the tube, resulting in permanent stricture. This occurs chiefly at the lower end of the tube, frequently also at the upper end. (See also inflammation of the stomach).

Perforating ulcers of the œsophagus are of occasional occurrence. They are analogous to those in the stomach. Instances are on record where they formed a communication with a bronchus (the left) and with the aorta. (*Vide* perforating ulcer of stomach).

Varicose ulcers sometimes occur at the lower end of the œsophagus. The varix is set up by some obstruction in the portal circulation, and it will be remembered that these œsophageal veins communicate with the portal system. Owing to the continual irritation of the mucous membrane over the varicose veins, caused by the food in its passage to the stomach, small rounded ulcers not uncommonly form. Sometimes a dilated vein is ulcerated into, and serious, perhaps fatal, hæmorrhage results.

TUMOURS.

Simple tumours are not of much importance either in the pharynx or œsophagus. **Polypi** are not uncommonly found in the upper part of the *pharynx*, but the majority of these are nasal in origin. They are usually myxomatous in nature, or myxo-adenomatous. Others grow from the connective tissue behind the pharynx and grow into the tube, being the direction of least resistance: these are fibrous tumours. In the *œsophagus*, fibroma, lipoma, and myoma have all been occasionally observed.

Malignant tumours are comparatively rare in the *pharynx*, but in the *œsophagus* they are of vital importance, CANCER is the most serious disease that affects the œsophagus, being a prolific source of stricture and consequent inanition. It occurs most frequently in males over the age of 40.

The variety of cancer which we have to deal with here is the **squamous epithelioma.**

Sites.—The most frequent site for a cancer of the œsophagus is in the lower third of the tube. It is also common in the middle third; sometimes it involves both these areas, *i.e.*, the lower two-thirds. A small proportion of cancers arise in the upper third.

Mode of growth.—The tumour starts at some point in the mucous membrane, producing first of all a nodular thickening, which in process of time comes to surround the tube, forming a prominent belt of several inches in depth. With the advance of the disease the œsophageal wall is gradually infiltrated until adhesions form outside. Meanwhile the lumen of the tube has been greatly narrowed, and ultimately no food can pass into the stomach. The outcome of this naturally is starvation and death, unless the patient is fed per rectum, or surgical interference be resorted to. In some cases the tumour ulcerates and may even slough away *en masse*, leaving the tube as large or larger than it was before. Such an issue is sometimes regarded as a natural cure, but a fatal result will usually follow from a secondary occurrence of the cancer. Moreover, in such cases, the muscular wall of the œsophagus will have been paralysed or destroyed, so that peristalsis can no longer be carried on; the effect of this may be that the food matter is retained in the tube as effectually as if an actual stricture existed, and the patient has all the symptoms of stricture. The disease usually proves fatal by perforation into the pleura or pericardium, more rarely by perforation into a vessel, causing fatal hæmorrhage.

Secondary growths arise in the mediastinal glands and various surrounding structures.

D. The STOMACH.

POST MORTEM CHANGES.

If death occurs when gastric digestion is in progress, and when consequently gastric juice is present in the stomach, *post mortem* changes are prone to occur in the contents of the stomach as well as in the gastric wall itself. In the first place the gastric juice exerts a morbid influence on the blood which is contained in the vessels of the wall of the stomach, and produces chemical changes in the hemoglobin, whereby the blood undergoes alterations in colour. As a result of this, the mucous membrane becomes stained of a red tint, which often changes to green and purple by decomposition changes. In the second place, it frequently happens that the digestive process, which normally influences only the contents of the stomach, is after death directed against the wall of the stomach, and the latter undergoes "*post mortem* digestion." This takes place generally in cases of sudden death, occuring soon after a meal, when there is an active secretion of gastric juice. It is favoured by a warm atmosphere. Under such circumstances the gastric juice attacks the de-vitalized mucous membrane, softening and dissolving it; the muscular coat and the fibrous coat may in turn be attacked, and the entire gastric wall be destroyed. As might be anticipated, these *post mortem* changes are observed only in such parts of the stomach as the contents have been in contact with. In ordinary cases where the body lies on its back, the posterior wall at the cardiac end is the affected part.

When the stomach is perforated in this manner, the contents commonly pass into the peritoneal cavity. But occasionally the perforation occurs near the root of the œsophagus, and then the contents may be evacuated into the pleural cavity.

The importance of these changes is twofold. Firstly, they are liable to mask other conditions which are evidences of actual disease; and secondly, they may themselves be mistaken for indications of corrosive poisoning. The latter contingency can be obviated by attention to the following points:—(1) The perforated wall is very soft, gelatinous, and fragile, the mucous membrane is almost liquefied, and the edge of the aperture is quite fringed with shreds of the tissue. (2) There is no sign of corrosive action in the mouth, pharynx, or œsophagus. (3) There is an absence of any staining, characteristic of such corrosives as sulphuric or nitric acid.

ATROPHY

Of the stomach is observed after chronic starvation. We shall afterwards see that the intestines suffer in a similar manner. The muscular coat and mucous membrane both become much thinned, and degenerative changes follow. In old age, the peptic glands become atrophied, and this is often accompanied by chronic catarrh.

DILATATION AND HYPERTROPHY.

The presence of any obstruction at the pyloric end of the stomach, preventing or hindering the passage of food, invariably causes dilatation of the stomach, which is compensated by a hypertrophy of the organ. Such obsruction is usually due to malignant stricture. The distension tells principally on the greater curvature, that being the most moveable part of the stomach, and so there is a downward extension of the organ, sometimes reaching to the lowest limit of the abdomen. The pyloric end, on the other hand, is the special seat of hypertrophy, since that part of the stomach is employed in forcing the contents through the pyloric orifice.

But the stomach dilates under other circumstances than those mentioned. A degree of dilatation is frequently got as a result of chronic gastric catarrh. Again, cases of distension have been observed that could only be accounted for by the assumption of a paresis of the muscular coat, whereby the wall was weakened and accordingly yielded to the internal pressure.

GASTRITIS,

Or inflammation of the stomach, presents several types.

Acute Gastric Catarrh is a very common condition. It frequently arises from errors in diet—including the use of irritating meat or drink, as well as excessive indulgence in otherwise harmless food. It is also caused by the action of phosphorus and other poisons, and it forms a symptom in the course of many fevers. Lastly, it very often results apparently from an ordinary chill, the result of exposure.

The **morbid phenomena** vary with the intensity of the inflammation. A slight catarrh does not materially differ in its manifestations from catarrh elsewhere. In severe cases of a common kind one observes an intense red swelling of the [redacted] membrane, often limited to a mu definite region, particularly the pyloric end and lesser curvature. The membrane is softened, and covered with viscid tenacious mucous, which is often discoloured by blood staining. At various points small capillary hæmorrhages can be detected, and in many cases there is considerable effusion of blood into the stomach. In consequence of the hæmorrhages there form (when the patient survives) small lesions termed "hæmorrhogic erosions," which will be described in connection with ulcerations of the stomach.

The inflammation induced by many irritant poisons is of a more diffuse character, and frequently involves the entire thickness of the gastric wall, leading to peritonitis externally. Quite a large quantity of blood may be effused into the stomach, discolouring its contents.

Certain chemical poisons produce characteristic changes in the stomach. Thus arsenious acid, in addition to producing an acute catarrh, imparts a bright yellow colour to the mucous membrane by being deposited in the form of the yellow sulphide. Corrosive sublimate causes a whitish wrinkling of the mucous membrane. Nitric acid produces a green or yellow discolouration, while sulphuric acid leaves a blackened and charred surface.

Chronic Gastric Catarrh is often a sequel to repeated acute attacks. It also affects individuals in whom there is some cause of venous congestion of the stomach, vizt., obstruction of the portal circulation, or long standing heart disease. But it occurs typically in inebriates as the direct result of alcoholic excess.

The **morbid phenomena** differ in degree ; in a well marked case they are very characteristic. As in the acute inflammation, so in this, the pyloric end is predominatingly affected. The mucous membrane is much thickened, and presents a warty or mammillated appearance. It is pigmented of a grey tint, the colour being especially pronounced around the warty eminences. A thick secretion of mucous covers the membrane. Hæmorrhagic erosions are found here and there : they appear as dark-brown spots, and when they exist in large numbers give rise to marked pigmentation. There is another and large ulcer commonly observed, vizt., a follicular ulcer, also called an alcoholic ulcer from its mode of origin. Both of these will be presently described. In addition to these phenomena, the wall of the stomach is much thickened and somewhat rigid.

Microscopically.—The mucous membrane is found enormously thickened, and it presents the usual characteristics of a chronic catarrh. At various points small cysts may be discovered : these are of the nature of retention cysts, and are simply dilated glands whose orifices have become obstructed by the thickened membrane. In the sub-mucous and muscular coats there is a great development of connective tissue, accounting for the thickened gastric wall.

ULCERATIONS.

I. HÆMORRHAGIC EROSIONS

Have been mentioned as occurring in catarrh of the stomach. They are simply pin-head ulcers, quite superficial and almost black in colour. They appear to be due to minute capillary hæmorrhages into the mucous membrane, and to the subsequent digestion of the damaged part by gastric juice. After healing they leave a dark-brown stain, and so a succession of them in time gives rise to striking pigmentation.

II. FOLLICULAR ULCERS.

(SYNONYM : ALCOHOLIC ULCERS.)

These occur as a phenomenon in chronic gastric catarrh. They are small, nearly circular, with sharp, undermined, vascular edges. They

involve the entire mucous coat, sometimes also the sub-mucous coat. As a rule these ulcers are multiple, and they develop in relation to the peptic glands.

III. PERFORATING ULCERS.

SYNONYMS: "SIMPLE," "CHRONIC," "CIRCULAR," "PEPTIC," "GASTRIC" ULCER.

This is a form of ulcer which is peculiar to the stomach, the first part of the duodenum, and the lower end of the œsophagus, and which often occurs as an independent lesion. It is of most frequent occurrence in the stomach, also fairly common in the duodenum, but very rare in the œsophagus. The morbid phenomena are practically the same in each case.

Perforating ulcer of the stomach is much more common in females than males, and occurs at a younger age in the former. Females are usually affected between the ages of 20 and 30, males between the ages of 30 and 40

Characters.—In an early condition one generally finds a single ulcer. Its appearance suggests that it might have been produced by the punching out of a circular piece of the mucous membrane. It is an inch or less in size, nearly round, and sharply defined. The edges are not undermined, but are quite vertical, and there is an absence of vascularity in the margins and base. At a somewhat later stage the ulcer is seen to be deeper, and the additional depth is of a smaller diameter than the older portion: to carry the original simile further, it seems as if the new loss of substance in the gastric wall had been brought about by the use of a smaller punch than in the first instance. At a later stage the ulcer is still deeper, and its diameter at the base is still narrower. In course of time these successive losses of substance deepen the ulcer to such an extent that the stomach becomes perforated. The ulcer has all the while been growing narrower and narrower at its base, so that the wall comes to have a terraced or stair-like appearance, and the perforation in the serous coat is often of minute size.

Although as a rule there is only one ulcer to begin with, as the condition becomes chronic, a second, sometimes a third, perhaps a fourth arises; and each passes through the same stages if the patient lives.

Sites.—Solitary ulcers of the stomach are almost always found on the posterior wall. The majority are situated towards the lesser curvature, a smaller number near the pylorus. Rarely is a gastric ulcer observed in the region of the greater curvature.

Effects.—During the progress of a gastric ulcer repeated hæmorrhages take place into the stomach; vomiting is frequently induced, and the vomited matter has the appearance of "coffee grounds." (That circumstance, together with the severe localized pain which the ulcer occasions, are the most important clinical points in the diagnosis of the condition.) A common result of the ulcerative process is perforation of the gastric wall. This usually terminates by the contents of the stomach being evacuated into the peritoneal cavity, where a fatal peritonitis is set up. But frequently, perforation issues more favourably. Thus it is not uncommon for adhesions to form between the wall of the stomach and the structure lying opposite the perforated spot. The pancreas is specially liable to be involved in this manner; the gland becomes indurated, and the ulcer burrows into it. The liver is less commonly involved; its invasion is prone to be followed by abscess formation. On the other hand, again, the ulcer after perforating the gastric wall may eat into some large vessel (usually the splenic artery or vein) and cause fatal hæmorrhage. It has just been stated that repeated hæmorrhage from the stomach is a symptom of gastric ulcer: these hæmorrhages are caused by the occasional erosions of small vessels in the stomach wall—frequently a branch of the gastric artery. Sometimes fatal hæmorrhage occurs in this way before the gastric wall is perforated.

Healing of a gastric ulcer takes place in a considerable number of instances, the process resulting in more or less cicatrization and deformity. In the case of an ulcer on a common site, the healing always produces a certain amount of puckering; and this is frequently aggravated by an irritative contraction in the circular muscular fibres at the spot, resulting in the development of a constriction across the organ, somewhere between the cardiac and pyloric ends. The appearance presented by the organ in such cases has led to the adoption of the name "hour glass contraction" as a descriptive term for the condition. As a rule one or more fresh ulcers develop on the cardiac side of the constriction. The healing of an ulcer situated at the pyloric end of the stomach sometimes leads to complete stricture of the organ in that region.

Causation.—There is much doubt and dispute as to the real exciting factor in the formation of these ulcers. It is admitted on all hands that the immediate cause of the loss of substance is a digestion by the gastric juice of a damaged part of the mucous membrane; but the origin of the damage is a debatable point. Virchow theorized that it arose from some interference with the arteries at the part, and suggested the probability of obstruction caused by an embolus. He based his theory on the shape of the ulcers. Experiments have shown the possibility of this mode of origin, but as a matter of fact one seldom discovers any such arterial obstruction in connection with gastric ulcers. Klebs was of opinion that the damage was due to a localized anæmia of of the gastric wall, resulting from spasm of the vessels. But this view is quite unsupported. Rindfleisch attributed it to venous congestion and hæmorrhagic infiltration of the affected spot, occurring as a sequel to thrombosis in the portal vein. But the portal vein is very often throm-

bosed and no gastric ulcer exists. A fourth view is that the ulcers are of inflammatory origin, but their appearance strongly negatives this.

Gastric ulcers occur very commonly in anæmic individuals, who usually suffer from dyspeptic symptoms. (Domestic servants may be specially instanced in this relation). Two factors probably come into play here. In the first place, the stomach may receive an imperfect supply of alkaline blood, and it seems that the alkalinity of the blood exercises a preservative influence against the action of the gastric juice on the lining of the stomach. In the second place, the gastric juice may be vitiated in some way, so that its digestive power is increased. In many cases it is found to be exceedingly acid.

Again, these ulcers are of frequent occurrence in phthisical patients. Here, also, anæmia has to be taken into account; but no doubt the ulceration is mainly a direct consequence of the action of the tubercular virus, and so these are to be regarded as **tubercular ulcers.**

Greenfield is of opinion that gastric ulcers are often **traumatic** in origin, occurring as a result of mechanical injury to the mucous membrane. His reasons for holding this opinion are two :—(I) The ulcers are frequently found in the stomachs of females who have been accustomed to tight lacing. (2) In one instance, a typical ulcer resulted from the incautious use of the stomach pump.

TUMOURS.

Various forms of **simple** tumours are occasionally met with. **Fibroma, myoma, adenoma,** and **lipoma** have been observed. They sometimes assume a polypoid form.

Malignant tumours. *Secondary* growths are now and then found on the stomach : the only one that need be mentioned is **lymphosarcoma.**

Of *primary* growths by far the most important is CANCER—important both on account of its frequency of occurrence and its results.

Sites.—The commonest position for a cancer is at the pyloric end. More than half of all cancerous tumours grow in that position. Next in frequency they occur in the lesser curvature, and in a smaller number of cases the cardiac orifice is affected. A certain percentage is found in other parts.

Varieties.—**Malignant adenoma** is perhaps the most common form. They may be found in any part of the stomach, and form a considerable proportion both of pyloric cancers, and cancers at the cardiac end. **Scirrhus cancer** is also common, and constitutes the bulk of pyloric cancers. It often extends to the lesser curvature and beyond. **Encephaloid** tumours are also of very frequent occurrence, and transitional forms between scirrhus and encephaloid are often observed. A scirrhus tends to assume the encephaloid type. **Colloid cancer** is fairly common, affecting chiefly the cardiac end. In many cases it consists simply in a colloid degeneration of another variety of tumour.

Characters and Effects.—These vary with the site. All forms of cancer commence in the glands of the stomach, involving at first the mucous membrane alone. But they invariably invade the other coats in course of time. Cancers at the pyloric end almost always cause stricture of the pylorus. This is not solely due to obstruction offered by the mass of the tumour, but is aided by a spasmodic contraction of the muscular coat in that region. The usual issue of this is death from inanition, often hastened by exhaustion from incapacity to take food. As mentioned on a previous page, the stomach suffers dilatation under such circumstances. Pyloric cancer sometimes remains limited to the starting point; in other cases it extends backwards into the stomach, producing great thickening of the wall, and often forming large fungus masses which tend to ulcerate and bleed. The hæmorrhage is never very extensive, however.

Cancers at the lesser curvature are prone to extend in a circular vertical direction round the stomach, and by their growth and contraction cause a constriction of the "hour glass" type.

Cancers at the cardiac end are often quite small and localized, but effectually prevent the entrance of food, causing rapid emaciation. They ulcerate at an early period, and tend to undergo colloid softening. They invade surrounding structures—the diaphragm and base of the lung especially—and on the whole prove more rapidly fatal than pyloric cancers.

Secondary growths are of very frequent occurrence in the liver. They usually start from the pylorus, which becomes adherent to the under surface of the liver, and that organ becomes the seat of a cancerous growth, usually encephaloid in character. Instead of a direct extension in this manner, the cancerous material frequently reaches the liver by way of the lymphatics. Colloid cancers produce secondary growths in the peritoneum much more readily than in the liver or lymphatic glands.

Hæmorrhage is a contingency to which cancerous growths are always liable. The encephaloid tumour, especially, is very prone to bleed. The bleeding as a rule is slight, and at frequent intervals, the blood being expelled into the bowel. Occasionally a large vessel is opened into by the ulcerative process, and fatal hæmorrhage occurs.

Death usually results from inanition, the consequence of obstruction at the pylorus or cardiac orifice. Fatal hæmorrhage, as just mentioned, is only an occasional occurrence. In very rare cases the fatal issue results from perforation of the stomach. Such perforation usually takes place into the transverse colon, forming a "gastro-colic fistula."

HÆMORRHAGE.

Bleeding from the stomach is a sign of various conditions, and

incidental mention has been made of the fact when considering these conditions. It will be useful to recount the latter briefly. (1) Chronic venous congestion—from disease of the heart or liver; (2) catarrh and ulceration; (3) perforating ulcer; (4) cancer. In addition, blood often collects in the stomach, when the hæmorrhage has occurred from a higher part of the alimentary canal. As instances of this, varicose ulcers and cancers of the œsophagus may be mentioned.

Blood may be evacuated from the stomach in one of three ways. It may pass into the duodenum, traverse the intestines along with the contents of the latter, and be ejected with the fæces. By that time it has become altered in character, and the stools consequently have a black tarry appearance. Or the blood may be vomited, a phenomenon designated by the term *hæmatemesis.* It is generally mixed with the contents of the stomach and darkened in colour by the gastric juice, hence the vomited matter often has the appearance of "coffee grounds," sometimes it is tar-like. When copious bleeding takes place into the stomach, the blood is apt to be ejected by vomiting there and then, and the blood in such a case has a perfectly fresh appearance.

E. The INTESTINES.

POST MORTEM CONDITIONS.

The intestine as well as the stomach is liable to exhibit *post mortem* appearances which may be misleading. The blood in the intestinal wall always gravitates to the lowest level of the coils; the colouring matter of the blood may then be liberated and stain the mucous membrane, a phenomenon which is apt to be mistaken for evidence of inflammation or gangrene, according to the degree of staining. Again, the coils of intestine lying in close proximity to the gall bladder may become stained with bile.

MALFORMATIONS.

There are several congenital anomalies to which the intestine is subject; some of them have been observed repeatedly. Occasionally, the intestinal tract has been found greatly shortened, presenting a simple S-like curve instead of a multiplicity of convolutions. Several instances of congenital narrowing or **atresia** of the gut have been met with. They are commonest in the duodenum, the lower end of the ileum, and the sigmoid flexure. Sometimes the gut is absolutely impervious, a condition incompatible with life.

The most important malformations, however, are those implicating the lower end of the intestinal canal, the rectum in particular. The chief of these is the surgical condition known as **imperforate anus.** This may assume one of several forms. Sometimes, there is simply no anal opening, the rectum terminating in a blind extremity. But generally the tube is deficient at its lower extremity, part of it being represented by a mere fibrous cord. In many cases, the condition is associated with stenosis of the sigmoid flexure.

A rare malformation is the formation of a cloaca, the rectal and vaginal canals having one common opening.

Amongst other malformations of the intestine, the commonest is that known as **Meckel's diverticulum.** This is due to the persistence of a fœtal structure—the omphalo-mesenteric duct. It is found on the small intestine at a point about three feet above the ileo-cæcal valve. (In the case of a child the distance is a foot or eighteen inches). It generally assumes the form of a finger-like projection several inches in length, attached to the free convex border of the gut. The pouch has a similar structure and is of the same calibre as the rest of the intestine, although sometimes it is much narrowed at the distal end and prolonged into a fibrous cord, which may be attached to the umbilicus. Occasionally, instead of a mere pouch, there is an absolute patency of the persistent duct, which extends from the intestine to the umbilicus, establishing at the latter situation a fistulous communication between the gut and the exterior. The significance of Meckel's diverticulum lies in the circumstance that, firstly, it is liable to be impacted by various foreign bodies, and secondly, that it may ensnare and strangulate a part of the intestine.

MECHANICAL INJURIES AND OTHER ACCIDENTS.

I. HERNIA.

This signifies "a protrusion of the intestine, or other abdominal organ into a sac formed by a prolongation of the peritoneum." The sac with its contents very often escapes through some weak point in the abdominal parietes, constituting an **external hernia.** On the other hand, sac and contents may be confined within the body walls, hence this condition is distinguished from the former by the appellation of **internal hernia.** External hernia, being a surgical condition of great importance, is considered at length in all surgical text-books; and so we shall limit ourselves to a study of the less common, though not unimportant, condition of internal hernia.

Diaphragmatic hernia is one of the commonest of internal herniæ. It consists in a protrusion of some portion of the abdominal viscera through an opening in the diaphragm—either one of the natural foramina or some abnormal passage. This is met with as a congenital condition, many of the viscera lying in the thoracic cavity. In the majority of cases, the hernia takes place through the left side of the diaphragm. It also occurs in acquired form, and is usually of traumatic origin. A severe crush has caused it, as also a fall from a height.

Retro-peritoneal hernia. This is a form of hernia in which a

1 Jejuno-duodenal
2 Sub-caecal
3 Fossa Intersigmoidea

portion of the intestine passes into a pre-existing pouch of peritoneum, the sac of the hernia lying behind the peritoneum of which it is an offset. There are three such pouches, each of which is named according to its position. We shall mention them in the order of their importance. (1) The **jejuno-duodenal** pouch lies on the posterior wall of the abdomen, between the third part of the duodenum and the aorta, and exists in about two-thirds of human individuals. Normally it will admit the terminal phalanx of the thumb, and a portion of the jejunum is liable to be ensnared in it. Some instances are recorded where the pouch was so distended that the entire intestine lay in its interior. (2) The **sub-cæcal** pouch is situated between the folds of the ascending mesocolon. It is present in about 30 per cent. of individuals only, and is an uncommon seat of hernia. (3) The **fossa intersigmoidea** is a pouch in the mesentery, lying between the two folds near the origin of the sigmoid flexure. Although it is the commonest of the pouches—being present in the great majority of persons—it is very seldom involved in a hernial protrusion.

Instances of internal hernia have been observed where the intestine passed through openings in the mesentery and in the great omentum.

II. TWISTS.

Twisting of the intestine is an important and not uncommon cause of obstruction of the bowel. It occurs almost invariably at the sigmoid flexure, a circumstance easily accounted for by the nature of the mesenteric attachments of that part of the bowel. Each end of the flexure is practically a fixed point, while the loop itself has great freedom of movement and is therefore liable to turn on its own axis. The result is that both extremities of the loop are compressed, one against the other, obstructing the entrance of fæces into the flexure as well as their exit from the tube. The obstruction is seldom complete, but the fæces are very liable to accumulate in the flexure, and enormous distension may ensue. The walls of the gut become thickened in consequence, chiefly due to hypertrophy of the muscular coat.

III. INTUSSUSCEPTION OR INVAGINATION.

These terms indicate a state of matters where a portion of the bowel comes to be contained within another portion. This originates mainly through excessive peristaltic action in one part, which gradually insinuates itself within a succeeding and more relaxed part.

Sites.—Intussusception takes place most commonly at the junction of the large and small intestine, the small gut passing through the ileocolic valve into the colon. Next in frequency one meets with invagination involving the small intestine alone: this generally takes place toward its lower end. Less frequently still, the large intestine alone is implicated: here as a rule the transverse colon passes into the descending colon. More rarely, the sigmoid flexure is concerned.

Intussusception is far more common in children than adults, a circumstance largely accounted for by the greater mobility of the intestines in the former. But further, *post mortem* examination of the bodies of children often reveals a condition of invagination in which the small intestine alone is concerned, and which without doubt occurs in the process of dying. It is a matter of common observation that dogs and other animals at the point of death, and even *post mortem*, exhibit violent peristaltic movement of the intestines. Such a phenomenon is not to be regarded as pathological, since of course it produces no symptoms nor after effects. The invagination can be reduced by the slightest traction on the bowel.

Mode of Occurrence.—It will be convenient to consider this in relation to intussusception at the ileo-colic valve—the most common site. The ileum by virtue of its peristaltic motion intrudes itself within the colon. Now it must necessarily happen that in the course of this process, either the ileum becomes everted (*i.e.*, turned inside out) or the colon inverted, depending on the question at whose expense the invagination takes place. As a matter of fact, the colon in the great majority of cases suffers invertion; the ileum, preceded by the ileo-colic valve, passes along the interior of the colon for a variable distance—the extent of invagination being limited by circumstances presently to be mentioned. In this way, the ileum does not actually pass through the ileo-colic valve, but carries that structure before it. But, in a small proportion of cases, the valve remains *in situ;* the ileum passes through the valve and becomes everted in the process.

When a piece of intestine is invaginated, a transverse section of the gut shows three tubes—an outer, an inner, and a middle one, named according to their relative position. These tubes, of course, are normally continuous. In the case under consideration, the ileum is the inner tube, constituting what is technically called the *entering layer.* The colon forms both outer and middle tubes, the former being known as the *sheath*, and the latter as the *outgoing* or simply the *middle layer.* The entering and middle layers are collectively known as the *intussusceptum:* the term "volvulus" has the same signification. The point where the entering layer is folded on itself to form the middle layer is called the *apex* of the intussusceptum. In consequence of the relations of the three layers, the mucous membrane of the sheath is in apposition with the mucous membrane of the middle layer, while the serous coat of the latter is in contact with that of the entering layer.

The progress of the intussusceptum along the sheath is due to the continued peristaltic action of the bowel. The extent of invagination is limited by the mesenteric attachment of the small intestine, aided by

the swelling which frequently ensues. But notwithstanding these restraints, it not unfrequently happens that the greater part of the small intestine is carried inside the colon. In consequence, the ileo-colic valve may reach the lower end of the rectum, when it can be felt through the anus. This is a fact of diagnostic importance. The mesentery of a necessity is dragged along with the intestine into the colon ; this circumstance causes the intestine to enter unevenly, and imparts a somewhat triangular shape to the lumen of the entering layer, when viewed in transverse section.

Effects. The first evil effect of the intussusception is to obstruct the circulation in the bowel : this results in swelling of the intestinal wall, frequently attended with hæmorrhage from the mucous membrane. The passage of blood per anum is therefore a diagnostic point of some consequence. This is very often followed by inflammation of the gut, manifested particularly on the serous surface, and leading to a localized peritonitis with adhesion between the entering and middle layers. In this way, the gut becomes practically strangulated (see next subject) and a fatal issue is the common termination. But sometimes there occurs a sloughing of the adherent layers at the proximal end of the invagination, resulting in their detachment and leaving the original entering layer in continuity with the sheath. The slough is then passed per anum, and the condition is naturally cured. However, the intestine besides remaining permanently shortened usually exhibits a degree of constriction at the seat of union.

Prolapsus Ani is a condition very often associated with invagination. It consists in the protrusion of a portion of the rectum through the anal orifice. This is usually the result of violent straining at stool, sometimes of inflammation. The lower part of the rectum, being firmly supported by its muscular attachments, is never wholly extruded : its mucous membrane is merely exposed. But the upper two-thirds is comparatively free, and may be invaginated into the lower portion and then prolapsed. The mucous membrane is exposed in each case, and is prone to inflammation and hæmorrhage.

IV. STRANGULATION

Of the intestine means the constriction of a portion of the gut, whereby its blood-vessels are obstructed and the part may die. This is a common sequel of (1) hernia, especially external hernia. The neck of the sac becomes constricted by the combined agency of bending of the gut, peristaltic movement, and accumulation of fæcal matter with its gaseous products, all these tending to force the intestine against the neck of the sac, and tighten the grasp of the latter. The vessels are in consequence obstructed—first the veins and then the arteries, and if the condition is not relieved gangrene results. Obstruction of the veins alone does not often give rise to gangrene. If the strangulation be relieved before gangrene has set in, usually a reactionary inflammation occurs: this is to be explained no doubt by damage to the vessels arising from the constriction. The inflammation may ultimately develop into gangrene.

(2) Intussusception often terminates in strangulation, as already mentioned. The necrosed gut usually becomes perforated, its contents are evacuated into the peritoneum and a fatal peritonitis results.

(3) Twisting of the gut sometimes leads to strangulation.

(4) A portion of intestine may become strangulated by the agency of Meckel's diverticulum, a fact previously noted.

(5) Some instances are on record where the vermiform appendix had formed a constricting band for the intestine, and caused necrosis of the latter.

(6) Lastly, a very common cause of strangulation is the existence of peritoneal adhesions, the result of inflammation. It is quite a common thing for these adhesions to form in the pelvis, and implicate a portion of the gut, which often becomes strangulated in consequence.

ATROPHY

Of the intestine occurs in cases of death from inanition. The intestinal wall becomes thinned, and in extreme cases so translucent that the contents are visible through it. A similar condition is often observed after death from wasting diseases.

HYPERTROPHY

Of the intestine sometimes occurs as a compensatory condition in cases of stricture of the gut. The fact was noted in connection with twists. The muscular coat becomes greatly thickened, and the mucous membrane usually shows a chronic inflammatory thickening which sometimes partakes of a polypoid character.

WAXY DEGENERATION

Is extremely common in the alimentary canal, occurring in most cases of general waxy disease. (It may be mentioned here that in the higher parts of the alimentary canal waxy disease is of little consequence. The condition is observed in the pharynx, seldom or never in the œsophagus and stomach.) The commonest site for the disease is the small intestine, particularly at its lower end. The large intestine is less often affected.

Characters.—To the naked eye, the mucous membrane appears anæmic and glossy, and it feels smooth, "almost like a piece of fine silk velvet." Iodine staining fluid differentiates the waxy points. In the small intestine, the capillaries of the villi are chiefly affected, and so one sees a great number of small brown points, corresponding to the vascular loops. The staining in the large intestine is more diffuse, for it contains

Free Haemorrhage into bowel due to.–

1 Intense Inflammation of fevers
2 Superficial burns of large extent
3 Ulceration
4 Strangulation
5 Malignant disease esp. of rectum & colon
6 obstruction of Superior Mesenteric Artery

no villi, and the entire capillary plexus in the mucous and submucous coats is implicated. The capillaries are prone to rupture, allowing of hæmorrhage into the bowel. In some instances follicular ulcers form on the mucous membrane. The **microscopic** characters are those of ordinary waxy disease. In addition, a certain degree of catarrh is usually present.

HYPERÆMIA.

The intestines are very often hyperæmic when examined *post mortem*, and this is commonly due to the mode of death. A real **venous congestion** is sometimes observed in cases of disease of the heart or liver: the mucous membrane then is swollen and catarrhal, perhaps pigmented of a livid hue. **Active hyperæmia** seldom occurs *per se*, it is almost always the precursor of inflammation. It is probable that a simple arterial hyperæmia leaves but little sign of its presence after death.

HÆMORRHAGE

Into the intestines results from a great many different conditions. Simple **ecchymoses** into the mucous membrane are found as a result of phosphorus poisoning, pernicious anæmia, and ulcerative endocarditis. They are most commonly met with in the duodenum.

Free hæmorrhage into the bowel may be due to any of the following conditions:—(1) The intense inflammation of fevers. (2) Superficial burns of large extent. The occurrence of hæmorrhage from the duodenum after extensive burns of the skin was first pointed out by Dr. Curling. Small ulcers are simultaneously produced in relation to Brunner's glands. The explanation of this phenomenon is unknown. (3) Ulceration of the intestine, as in typhoid fever, dysentery, and cholera. (4) Strangulation of the intestine. (5) Malignant disease, particularly of the rectum or colon. (6) Obstruction of the superior mesenteric artery. This sometimes gives rise to extensive hæmorrhage from the portion of bowel corresponding to its distribution. The occurrence is attributed to collateral hyperæmia in an area which has suffered damage from a defective supply of blood.

Blood when passed by the bowels is never of a red colour. It always becomes blackened by the action of the intestinal secretion and gases, and imparts a tarry appearance to the stools. This leads to the condition of **melœna**. It may be simulated by the use of certain drugs, including iron and bismuth. Microscopic examination is the best distinguishing test.

ENTERITIS,

Or inflammation of the intestine, occurs in a great variety of forms. It may constitute a disease in itself, or exist as a symptom in many other diseases. Under the above heading we shall consider only the simple inflammations.

I. CATARRHAL ENTERITIS.

A. IN THE SMALL INTESTINE.

Acute catarrh is not common in the small intestine, and is chiefly met with in children. It occurs as a result of eating various noxious and indigestible substances which lie in the bowel and cause irritation. Sometimes it follows exposure to cold, and it also occurs in the course of certain fevers.

The **morbid phenomena** are not very distinctive. The ordinary appearances of catarrh are observed, hyperæmia being most marked on the valvulæ conniventes. In some instances the solitary glands are particularly swollen and project like small shot upon the surface of the mucous membrane: this phenomenon receives the special name of *psorentery*. The Peyer's patches may also be swollen, simulating commencing typhoid. Occasionally the solitary glands ulcerate, giving rise to follicular ulcers. The contents of the intestine are usually fluid, and mixed with mucus.

Chronic catarrh of the small intestine is a common occurrence. It is a result either of chronic venous congestion or of alcoholism. Sometimes it is a sequel of the acute inflammation.

The **morbid phenomena** generally resemble those of the acute variety. The mucous membrane is thickened and pigmented. The pigmentation often takes the form of small dark spots, many of which are disposed circularly around the swollen solitary glands.

B. IN THE LARGE INTESTINE.

Acute catarrh may occur here under similar conditions to its occurrence in the small intestine. The appearances vary to some extent on account of the different structure of the gut.

Chronic catarrh of the large intestine is a very common affection in the adult. It is generally due to the irritation of fæcal accumulations in the bowel, and therefore manifests its effects in those parts of the bowel where stagnation of fæces is most prone to occur, viz.,—in the cæcum with its vermiform appendix, and in the upper part of the rectum.

The **morbid phenomena** are not specially characteristic. The mucous membrane becomes greatly thickened, cysts form in it and polypoid outgrowths sometimes spring from its surface. The membrane is often pigmented. The gut may be considerably dilated and its muscular wall thickened. Then in addition, it is common to find follicular ulcers, resembling those in the stomach. They are about a quarter-of-an-inch in size, and develop from the solitary glands. They generally penetrate the mucous membrane only. In some cases, the ulceration spreads so that neighbouring ulcers coalesce at points, giving rise to a worm-eaten appearance in the gut. When the small ulcers

heal they leave a minute rounded or oval cicatrix with pigmented edges. These can be detected by holding the intestine between the eye and a bright light, also by floating the gut on water.

(Follicular ulcers in the intestine are sometimes got as a result of waxy degeneration and Bright's disease).

II. FIBRINOUS ENTERITIS.

This is a rare disease, but it is occasionally observed in the large intestine. It has occurred as the result of intense irritation caused by fæcal concretions and foreign bodies.

Characters.—On the surface of the gut one finds a layer of fibrinous exudation—often in patches—which, when stripped off, brings the mucous membrane with it. In many cases it entirely replaces that membrane. The exudation is usually blood-stained. The entire wall of the intestine shows inflammatory thickening, and not uncommonly there is localized peritonitis outside the wall. Sometimes fibrinous casts of the intestine are expelled per anum: these consist of great numbers of altered epithelial cells mixed with mucus.

III. PHLEGMONOUS ENTERITIS.

This, like the former, is a rare condition. It consists in the presence of suppuration beneath the mucous membrane. In cases of chronic Bright's disease and ulcerative endocarditis, one occasionally discovers small points of suppuration in the sub-mucous coat. These generally rupture the mucous membrane and form small ulcers.

In some instances a diffuse suppuration has been observed in the sub-mucous coat. This has been described as **mycosis intestinalis**, and is supposed to be due to the agency of *anthrax bacillus.*

GANGRENE

Of the bowel is usually a result of strangulation—the sequel, in particular, of hernia or intussusception. In rare cases the intense inflammation of typhoid fever or dysentery leads to gangrene of portions of the gut. When a portion of intestine is nipped in a hernia, it becomes first of all dilated in consequence of paralysis of the muscular coat. The wall becomes greatly swollen and congested, exhibiting a deep red colour, which soon becomes livid. A quantity of blood is extravasated into the bowel as well as on its peritoneal covering. The wall then begins to turn green at points, afterwards black, until the entire hernia is of a uniform dark colour. It has by this time become necrosed, and sloughing is apt to follow, leading to perforation of the gut and escape of the intestinal contents, the issue of which is fatal peritonitis. Sometimes the very intensity of the inflammation destroys the vitality of the part so rapidly that no sloughing takes place, and a simple local peritonitis is set up around.

Microscopically, the inflamed mucous membrane commonly exhibits a catarrhal exudation, but in rare cases the exudation is distinctly fibrinous.

INTESTINAL DISEASE due to SPECIAL POISONS or FEVERS.

I. ASIATIC CHOLERA.

This is an epidemic disease, well known in tropical and many temperate climates. It is characterized by the discharge of very watery stools, which closely resemble rice water in appearance. The fluid element of the stools is derived partly from the blood and partly from a great hyper-secretion of the intestinal glands, while the flaky masses that float in it consist of large numbers of leucocytes, aggregated in a thick mucus.

Post mortem, the blood is found thick and tarry, owing to the withdrawal of such large quantities of fluid. In the intestine slight lesions are observed. The lower part of the small intestine exhibits "a remarkable rosy injection of the vessels of all its coats, so that a red appearance is visible in the serous coat whenever the body is opened." Sometimes there are minute ecchymoses under the mucous membrane. The lymph follicles are generally swollen and prominent. Occasionally there is some superficial necrosis of the mucous membrane.

Koch, in the course of researches in India, discovered an organism, which is now recognised as the exciting cause of cholera. It exists in the shape of a very short curved rod, not unlike a comma, and Koch applied to it the name of *comma bacillus.* The organism can be found in the stools, in the glands of the intestine, and in the mucous membrane generally. In grows into longer spiral threads, of which the short rods are probably segments. In water the bacillus possesses great vitality, but it is readily destroyed by drying. A low temperature is also fatal to it.

II. TYPHOID OR ENTERIC FEVER.

In this disease very characteristic lesions are found in the intestine. The small intestine is especially affected. "The disease has usually its point of greatest intensity at the lower extremity of the ileum, and it is often possible to see various stages of the process in proceeding from above downwards through the small intestine till it culminates close to the ileo-cæcal valve." The morbid changes specially involve the adenoid structures, including solitary glands, Peyer's patches, and mesenteric glands. The large intestine is affected in one out of every five cases on an average (Greenfield).

Morbid Anatomy.—A tolerably definite series of changes occurs in the intestine, extending over a period of three or four weeks, and the

phenomena are often described as occurring in stages of a week's duration. Thus, during the *first week*, the Peyer's glands along with the solitary glands are congested, swollen, and prominent above the surface. There is sometimes hyperæmia of the whole mucous surface, but this is not constant, indeed the Peyer's glands may be affected alone. In the *second week*, a certain number of the glands may subside and return to normal. But the majority undergo further enlargement, and the mucous membrane generally is now uniformly swollen. Many of the patches become confluent. They begin to exhibit some pallor, and then a process of necrosis commences. It starts at isolated points in the Peyer's patches, forming minute ulcers. By the extension of these, a large slough comes to cover the patch in great degree. The necrosed tissue is quite soft and coloured yellow from bile staining. (Biliary colouring matter always stains dead tissue). In the course of the *third week*, the slough separates, being practically digested away in fragments, and when separation is complete an ulcerated surface is exposed constituting a typical "typhoid ulcer." These ulcers seldom involve an entire Peyer's patch: they generally cover portion of a patch, and extend for a variable distance into the surrounding mucous membrane. Their long axis frequently coincides with the long axis of the gut. They have, as a rule, soft vascular undermined edges, and their floor is usually constituted by the muscular coat of the intestine. At the time of separation of the slough, a certain amount of hæmorrhage occurs, but the walls tend to become less vascular after the ulcer is once formed. The mesenteric glands are enlarged and softened.

Results.—Most commonly the ulcers undergo a process of healing. The first step in the healing process is a falling in of the edges, which become adherent to the floor. Then the ulcer heals like an ordinary granulating wound. The site of a healed ulcer is afterwards indicated by a smooth, depressed, pale or slate-coloured area, replacing part of a Peyer's patch, and usually surrounded by a zone of pigmentation. Cicatrized ulcers seldom give rise to any stricture of the bowel. When stricture does occur, it is usually the result of a localized peritonitis over the region of the ulcer.

Hæmorrhage takes place from the ulcer during the separation of the slough, as already mentioned. It is due to the erosion of vessels in the floor of the ulcer.

Occasionally, a typhoid ulcer extends deeply into the wall of the intestine, and perforation may result. Such an extreme issue as a rule takes place somewhat slowly, allowing time for peritoneal adhesions to form and so mend the rent. But it now and then happens that perforation occurs quickly, and the contents of the gut are evacuated into the peritoneum, setting up an acute peritonitis which proves fatal.

Ulceration in the *large intestine* differs in its characters from the more common lesion which has been considered. The inflammation chiefly affects the cæcum, where ulcers are formed by the confluence and subsequent necrosis and sloughing of the solitary glands. The ulcers generally have their long axis directed across the gut, and in other respects the condition is very much like dysentery.

A secondary follicular ulceration occurs sometimes, affecting both large and small intestines. It is very wide spread, and the ulcers are small and circular.

DYSENTERY.

This is a disease of great frequency in tropical climates: it is uncommon in this country. Its origin is doubtless to be traced to some specific organism, but how the organism enters the system is unknown. The disease manifests itself in an inflammation of the bowel, usually limited to the lower part of the large intestine—thus differing prominently from typhoid. The inflammation is most intense at the flexures, where fæcal matter is prone to accumulate, and extends a variable distance up the bowel—seldom further than the descending colon. There is an *acute* and *chronic* form of the disease.

I. ACUTE DYSENTERY.

Characters.—In the milder cases, the inflammation partakes of a catarrhal nature. The mucous membrane becomes covered with a slimy mucus, often tinged with blood. When this is removed the membrane is seen to be red and swollen, and thrown into folds, on the summit of which the hyperæmia is most marked. The solitary glands stand out prominently as flat swollen patches. These phenomena gradually become aggravated, until the folds of mucous membrane begin to suffer necrosis and exhibit small points of ulceration. These ulcers are at first superficial, and occupy the summit of the folds; they have hyperæmic edges, and the base is covered with a slough which is brown in colour from bile staining. Some fibrinous exudation is usually thrown out on the mucous membrane. By degrees the ulceration extends over wide areas of the gut, and large flakes of mucous membrane slough away. The bowel becomes gangrenous and discoloured, and the contents, which are deeply blood-stained, possess a very foul odour.

Microscopically.—All the usual appearances of intense catarrh are present. The ulcers are seen to have originated largely in the solitary glands. The contents of the bowel are composed of (1) necrosed epithelium, (2) mucus, (3) fibrin, (4) leucocytes, and (5) blood, in addition to fæcal matter. This mass of material is usually found in a state of decomposition, emitting a somewhat peculiar odour almost diagnostic of the condition.

II. CHRONIC DYSENTERY.

This usually supervenes on the acute form. The mucous membrane

exhibits large, pale, unhealthy ulcers with sloughing edges. The intestinal wall is thickened, unless where these ulcers exist, and generally is adherent to surrounding parts.

Results of dysentery.—In mild cases complete healing may occur, even after small ulcers have formed. But the chronic ulcers have little disposition to heal: they remain open and constantly emit a discharge. In some cases they do heal to a greater or less extent, with the result that cicatrices form, which exert an injurious traction on the intestinal wall. The gut may be contracted in its length, or the transverse diameter may be diminished and the bowel obstructed. A healed ulcer leaves a slight depression with pigmented edges: it can be advantageously seen by examining the gut by transmitted light. In rare instances acute ulcers perforate the bowel, and cause abscess formation or peritonitis according to the point of perforation.

In association with dysentery one frequently finds abscess of the liver. The latter is in many cases caused by the carriage of septic emboli from the intestine.

TUBERCULOSIS OF THE ALIMENTARY CANAL.

Tuberculosis is set up in the alimentary canal by the introduction of material containing tubercle bacilli. A large proportion of cases are secondary to tubercle in the lung, the infection being derived from swallowed sputum.

(1) The Tongue.

Minute tubercle granulations are found on the tongue in cases of general tuberculosis.

(2) The Pharynx

Is frequently the seat of small tubercular ulcers, superficial in character. They are often found coincidently with similar ulcers in the larynx.

(3) The Stomach.

Tubercular ulcers of the stomach are met with in cases of acute tuberculosis in children. There are usually two or three, quite similar to those in the intestine. Sometimes chronic ulcers are found.

(4) Small Intestine.

The chief lesion here is an ulcerative process which is generally of a chronic nature. It takes place in the lower end of the ileum as a rule. There is, to begin with, a series of inflammatory changes in the Peyer's glands and solitary glands, closely resembling the phenomena of typhoid. The swollen glands soon ulcerate and give rise to irregularly shaped ulcers, around which tubercle granulations are seen invading the mucous membrane. The ulcers gradually extend, and they very often acquire an oblong, somewhat rectangular shape. Their long axis runs transversely to the gut, and they sometimes extend completely round the bowel. The base of the ulcer consists of infiltrated and thickened mucous membrane, so that the muscular coat is not exposed. The edges are not undermined ; the margins are thickened and nodular, often terraced, and generally hyperæmic. The peritoneal covering over the ulcers is infiltrated with tubercles, which are discernible as yellow lines along the lymphatics, and the mesenteric glands are enlarged.

(5) Large Intestine.

The large intestine is not so often affected as the former, but the changes produced are much the same, implicating the solitary glands.

Results of Intestinal Ulceration.—Tubercular ulcers very rarely perforate the bowel. But when they heal, the cicatrization is so extensive that stricture of the gut is not uncommon.

SYPHILIS OF THE ALIMENTARY CANAL.

It has been mentioned that syphilis very often manifests its effects in the mouth and pharynx. The œsophagus and stomach are seldom affected. The only part of the bowel liable to be affected is the rectum and lower part of the large intestine. The disease causes an irregular thickening of these parts, and this is usually observed in the case of women who have syphilitic eruptions on the vagina.

TUMOURS.

Simple.—**Myomata** and **lipomata** are both found. The latter sometimes take the form of enlarged appendices epiploicæ. On the mucous surface **fibrous** and **mucous polypi** sometimes arise as the result of prolonged irritation.

Malignant.—**Lymphosarcoma** has an especial tendency to grow in the intestines. It sometimes forms small swellings, which ulcerate ; at other times it produces a diffuse infiltration. The most important morbid growths, however (as in the œsophagus and stomach), are CANCERS, of which we find several varieties. **Scirrhus** is uncommon, though occasionally found in the rectum. **Colloid** cancer may develop from any part of the intestine, and it frequently invades the wall and extends to the peritoneum. The commonest variety is **malignant adenoma.** It grows especially in the large bowel, and particularly in the cæcum, sigmoid flexure, and rectum. In the latter position the tumour constitutes "malignant polypus" of the rectum. The growth infiltrates the wall of the bowel in a circular manner, and ulceration quickly ensues. In the various coats of the intestine there is a new formation of connective tissue, and the consequent contraction very often leads to stricture of the bowel. "In fact, if we except herniæ, this is probably the most frequent cause of intestinal obstruction."

DISEASES of SPECIAL PARTS of the INTESTINE.

I. THE DUODENUM.

DUODENITIS.

This signifies a catarrh of the duodenum, and it is generally associated with catarrhal inflammation of the stomach. The condition is of little moment in itself, but becomes of great significance from the fact that the inflammation often extends into the common bile duct, causing obstruction of that tube, and leading to "catarrhal jaundice" (*v.* page 221).

SIMPLE PERFORATING ULCER.

The occurrence of this lesion in the duodenum has been already mentioned. There are usually several ulcers, situated close to the pylorus. They are almost identical with those in the stomach, but they seem to have a greater tendency to perforate than the former.

ULCERATION FOLLOWING BURNS.

It has been observed that in cases of severe burns terminating fatally the duodenum is the seat of numerous small ulcers, from which considerable hæmorrhage occurs. The ulcers are found on the mucous membrane of the first part of the duodenum, and arise in connection with Brunner's glands. The relation of the burn to the occurrence of this ulceration and hæmorrhage has not been explained.

NEW GROWTHS.

The duodenum is specially liable to be invaded with growths arising in the head of the pancreas. The anatomical relation of the parts naturally accounts for this.

II. THE CÆCUM.

TYPHLITIS,

Meaning inflammation of the cæcum, is a very common result of the retention of hard fæces in that portion of the bowel. A stricture of the gut beyond the cæcum often sets up typhlitis; and a mild degree of inflammation sometimes follows exposure to cold. The condition may be catarrhal or ulcerative : in many cases there is a degree of membranous exudation.

A very common sequence of malignant stricture of the colon is the occurrence of follicular ulcers in the cæcum : these generally perforate the bowel and cause peritonitis, which ends fatally.

Peri-typhlitis is a name used to designate inflammation in the cellular tissue around the cæcum. In most cases it arises by extension from the cæcum, probably by the gradual perforation of the wall by follicular ulcers. This usually occurs in the posterior wall, and it may lead to abscess formation behind the cæcum.

III. APPENDIX VERMIFORMIS.

CONCRETIONS.

A very common accident is the impaction of fæces or other foreign body in the vermiform appendix. Masses of fæces so impacted become inspissated, and frequently assume the form of cherry or date stones. The continual irritation of such hard masses sets up inflammation of the appendix, generally followed by ulceration and perforation. In most cases adhesions form around the appendix, and so the perforation primarily results in abscess formation. These abscesses may recur several times, and at length one ruptures into the peritoneum and a fatal peritonitis results.

DILATATION.

When the orifice of the appendix becomes closed—as it may be from catarrh—the tube dilates under the pressure of its accumulating secretion, forming a real retention cyst. The mucous contents generally become serous after a time.

DISEASES OF THE LIVER.

MALFORMATIONS.

The liver has been found absent once or twice in the newly-born fœtus, and instances of defect in one or other lobe are occasionally met with. Sometimes there are one or more small accessory livers.

The liver is liable to changes of position. A pleuritic effusion which depresses the right side of the diaphragm necessarily causes a downward displacement of the liver. The organ is sometimes elevated by the presence of fluid or tumours in the abdomen.

Acquired deformity of the liver is caused by the wearing of tight belts in the male and tight lacing in the female. The organ is more or less flattened, and a furrow is found running transversely across the upper surface of the right lobe. The furrow may become so deep that the organ is practically divided, the two portions being held together by the much thickened capsule.

HYPERTROPHY

Of the liver, pure and simple, rarely occurs. It is described as occurring in gout and diabetes, but in both these conditions further changes are to be found which do not correspond with the true definition of hypertrophy.

ATROPHY

Of the liver is very common. It occurs as a simple change in old age: the organ is smaller, the capsule is wrinkled and lacks its natural

gloss, the tissue is flabby though tough. Atrophy is sometimes caused by the pressure of tumours, and by peri-hepatitis.

CLOUDY SWELLING

Of the liver occurs under all conditions involving high temperature.

Characters.—In the early stage, the liver is hyperæmic, but as swelling becomes more marked the organ exhibits pallor.

Microscopically, the lobules appear denser than normal, and at the periphery the hepatic cells seem to encroach upon the capillaries and take their place. The cells are intensely granular and rounded instead of being polygonal. The nuclei do not stain with ordinary reagents, although one here and there may be observed to stain very deeply. In the portal canals, there is an increase of small round cells.

The condition may end in (1) resolution, (2) fatty degeneration, or (3) acute parenchymatous necrosis, when the whole cell becomes broken up into minute granules and disappears.

FATTY DEGENERATION

Of the liver occurs mainly in (1) wasting diseases, *e.g.* cancer and Addison's disease, (2) fevers, as a sequel to cloudy swelling, and (3) phosphorus poisoning.

Characters.—The organ in a typical case is smaller than normal, and exhibits a yellowish pallor. It is flabby and friable. In the extensive and rapid fatty degeneration produced by phosphorus, the liver is generally above the normal size and exhibits a bright yellow colour. Minute hæmorrhages are frequently present, both under the capsule and scattered through the organ.

Microscopically, the change is seen to affect all the zones of the lobule, the outer one perhaps more than the rest. The hepatic cells are diminished in size and filled with minute fat droplets, among which the nuclei can be distinguished. The bile ducts are frequently involved.

FATTY INFILTRATION.

The liver may become the seat of fatty infiltration in consequence of excessive indulgence in fat forming food. Very often the condition results from chronic alcoholism and phthisis. Sometimes it occurs in combination with other disease of the liver, such as waxy disease, cirrhosis and chronic venous congestion.

Characters.—The organ is enlarged and rounded, the capsule being stretched. Its weight is increased while the specific gravity is diminished. The tissue is soft, pits on pressure, and is very friable. When making a section, the knife becomes greasy, and if one scrapes the surface of section the knife carries with it minute fatty particles. Each individual lobule exhibits a pale yellow margin ; sometimes a yellow tint pervades the entire lobule. It should be noted that the yellow colour is often obscured by congestion in the central vein.

Microscopically.—In an ordinary simple case, the change is limited to the outer zone of the lobule. (It is in this position that fat is first deposited during digestion.) The liver cells in that area are swollen and contain large fat droplets, which exist at the expense of the protoplasm of the cell. The pressure exerted by the accumulated fat destroys the protoplasm and pushes aside the nucleus, hence the latter is generally found at the periphery of the cell. In very advanced cases, all three zones are invaded, diminishing the capillary space, but the condition never gives rise to sufficient obstruction to cause dropsy. Nor does it ever give rise to jaundice.

WAXY DEGENERATION

Of the liver is, in this country, associated with certain cachectic conditions which produce waxy disease in other organs, vizt. (1) phthisis, (2) chronic suppuration, particularly connected with bone, and (3) syphilis.

Characters.—In a well marked case, the entire liver is affected. The organ is enormously enlarged, and its configuration viewed from above is somewhat square shaped. The weight is much increased, as is also the specific gravity. The edges of the organ are thick and rounded: the recognition of this fact in regard to the anterior edge is a point of clinical importance. The tissue is hard and lacks its usual elasticity. The capsule is necessarily much stretched, rendering it thin and transparent. The surface of the organ is pale, and exhibits a colour resembling bacon or smoked salmon. On section, the edges remain sharp. A thin slice of liver tissue is translucent. Examination of the surface of section frequently shows that the lobules are uncommonly distinct. This is accounted for by the existence of fatty infiltration in the outer zone. The middle zone of the lobule (which is broader than the peripheral zone) has a glistening translucent appearance, while the central zone is dark. Iodine stain imparts a mahogany brown colour to the middle zone, indicating the site of the waxy change.

Microscopically.—The first signs of waxy degeneration are usually recognized in the intermediate zone of the lobule, where the small branches of the hepatic artery are affected. The process extends into the capillaries, and the outer zone of the lobule is gradually invaded. The fibrils of connective tissue in the portal canals then become affected. Since the change leads to an increase in size of the capillaries, these come to exert undue pressure on the surrounding liver cells, which suffer atrophy often with fatty degeneration.

In tubercular cases, one constantly finds tubercle granulations in the waxy parts. In cases of syphilis, gummata or cicatrices are frequently observed; cicatricial contraction may render the organ smaller.

ACUTE YELLOW ATROPHY

Is a disease of the liver which always terminates fatally. It usually affects the female sex, and in about half the number of cases it starts during pregnancy—generally before the 6th month. In some cases it occurs as a sequel to catarrhal jaundice or malarial fever, and in others it originates in pyæmic conditions. The exciting cause of the disease is unknown, but very probably it is of the nature of a morbid poison circulating in the blood.

Clinically, the disease is characterized by jaundice, vomiting, certain nervous phenomena and scattered hæmorrhages; terminating in coma, with low temperature. A pregnant patient invariably aborts before death takes place. Examination of the urine shows the existence of crystals of leucin and tyrosin.

Morbid Anatomy.—If the organ could be seen at the beginning of the process it would prove to be enlarged. This enlargement is temporary and soon disappears. After death the liver is found very much wasted, the capsule in consequence being excessively wrinkled. The organ is soft and flabby, but is rather tougher than the normal liver. On section, the surface exhibits an intense canary yellow or brownish yellow colour, variegated with some patches of red, which are disposed at irregular intervals. These red parts are not so soft as the bulk of the organ, and they are mainly related to the larger blood-vessels.

Microscopically.—In an early stage, there is cloudy swelling of the liver cells. Quickly succeeding this, there is some increase in the small round cells in the portal spaces: and almost simultaneously there is an acute parenchymatous necrosis in the hepatic cells, while the inflammatory process continues to increase at the margin of the lobules. The bile ducts are rendered more visible as the change proceeds: they appear to be more numerous also, and possibly there actually is a new formation of bile capillaries. As the liver cells break down and become fatty, the congestion of the capillaries increases, and a small cell exudation extends from the periphery of the lobule between the rows of liver cells. In consequence of these inflammatory processes the liver cells become atrophied and disappear to a large extent. If one now examines a lobule situated in the yellow part of the organ, the hepatic vein is seen in the centre, and radiating out from it are rows of blood and bile capillaries, between which the debris of liver cells can be recognized. Crystals of leucin and tyrosin, as well as brown rhombic crystals of unknown origin, are observed among the cells. The yellow colour of the tissue is due to bile staining. The red patches are in a more advanced condition than the rest, and in them the hepatic cells have quite disappeared.

Acute yellow atrophy of the liver is always accompanied by intense catarrhal changes in the kidney. In the early stage of the disease micrococci have sometimes been found in both organs.

CHRONIC VENOUS CONGESTION.

This occurs in the liver from mechanical causes involving obstruction to the venous return from the organ. In the case of a general congestion, the commonest cause is cardiac embarrassment, arising either from valvular lesions or disease of the lung. Sometimes the seat of obstruction is the inferior vena cava, which suffers from the pressure of tumours or aneurisms. The condition of the liver is shared by the various organs connected with the portal system, and various secondary symptoms also develop—such as hæmorrhoids and ascites.

A localized congestion results from pressure of a tumour or cyst, or inflammatory growth upon a branch or branches of the hepatic vein within the liver.

Characters.—A prolonged venous congestion of the liver leads to the occurrence of "cyanotic atrophy" of the organ, or "nutmeg liver," names which are each descriptive of the condition. The first thing to occur is a great distension of the hepatic veins with their capillaries, and the organ suffers consequent enlargement. On section, it is unduly red, and a large quantity of blood exudes from the distended veins. On minute examination, the centre of the lobule exhibits a darker red colour, while the middle zone is yellowish from bile staining, and the peripheral zone is pale and fatty. The variegation produced by these colours simulates the aspect presented by a section of a nutmeg. The capsule very often has villous processes attached to its exterior: these are a result of the ascites which frequently exists pari passu with the hepatic congestion. At an advanced stage of the condition the liver is atrophied. The surface is of a deep red colour, and often presents irregular elevations and depressions, resembling a cirrhotic liver. On section, each lobule is seen to be of a nearly uniform dark-red colour, with the exception of a narrow margin of yellow due to fatty changes. The organ is now in the condition of "cyanotic" or "red atrophy."

Microscopically.—In a nutmeg liver the central vein is seen to be dilated; its surrounding tributaries are distended and tortuous. The walls of the vessels are thickened. Between the dilated capillaries the liver cells are first compressed and rendered more angular: the result of this pressure is to cause atrophy of the cells, which soon become small and granular, and ultimately break down. The hepatic cells may entirely disappear from the central part of the lobule in this manner; and that region is occupied by a cavernous structure, made up of the dilated capillaries, whose contiguous walls have coalesced to form fibrous bands bounding spaces filled with blood. This extreme atrophy of liver tissue, in conjunction with the colour of the organ, has led to the

use of the term "red" or "cyanotic atrophy." Before the liver cells have disappeared, they may be observed to contain an excess of pigment granules. The source of this pigment is doubtful; probably it originates from a proliferation of the normal pigment in the cell. In the remains of cells that still exist towards the periphery of the lobule this pigment is very evident. Sometimes, in addition to these typical changes, there occurs a proliferation of the interlobular connective tissue, and this imparts to the liver in slight degree the character of cirrhosis.

HEPATITIS,

Or inflammation of the liver, occurs in both acute and chronic form.

I. ACUTE HEPATITIS.

A **simple** inflammation of the liver occurs as a result of various specific fevers—typhoid, for example. It is characterized by a great exudation of leucocytes into the portal spaces—so great sometimes as to give rise to the expression "typhoid new growths" in describing the condition.

Suppurative hepatitis.—Various forms of abscess occur in the liver.

(1) **Tropical abscess** of the liver is met with only in hot climates, and is therefore unknown in this country. In most cases it is associated with dysentery, and doubtless there is a causal connection between the two conditions, septic material being conveyed from the ulcerated intestine to the liver. But tropical abscess is often found when there is no dysentery, and the origin of the abscess under these circumstances is a source of conjecture. The abscess is usually solitary, starts deep in the liver substance, and grows towards the surface, expanding the liver as it advances. When it reaches the surface, it may rupture into the peritoneum, or form adhesions with the abdominal wall or viscera. In the latter case, the abscess may ultimately rupure externally or into an adjoining viscus, *e.g.*, the lung or colon. The contents of such an abscess are broken down liver tissue, with pus and blood; they possess a pink colour and fœtid odour. The walls are ragged and exhibit no pyogenic membrane. *Microscopically*, the condition is observed to commence with thrombosis of some branch of the portal vein, and large numbers of micrococci can be detected in the vicinity.

After the abscess ruptures, it continues to discharge—often for a very long time—and healing does not as a rule take place. In some instances the discharge ceases, the abscess walls shrink, and a cicatrix forms, leaving an indentation on the liver. But a tropical abscess does not always reach the surface; sometimes the contents dry in and caseate, afterwards calcifying, and the condition is naturally cured.

(2) **Pyæmic abscess.**—The liver is not a common site for pyæmic abscesses, and they only infect that organ under special circumstances. Thus a wound of the skull penetrating the outer table, if allowed to become septic, may set up pyæmia attended with abscess formation in the liver. Or abscesses may result from septic ulceration in the intestinal canal (*e.g.*, in dysentery), also from phlebitis of the portal vein.

Characters.—The abscesses are multiple and of small size. They have ragged walls and contain putrid pus. Around each is a zone of intense congestion. When phlebitis is the origin of the condition, the abscesses may be observed to correspond to the distribution of the particular vein. The vein is found to contain decomposed clot, resembling pus. The condition usually terminates fatally by the occurrence of peritonitis.

(3) **Cystic abscess.**—This results from suppuration in a hydatid cyst. Such abscesses are sharply defined, and have smooth walls : the true lining membrane of the cyst is destroyed.

(4) **Tumours** of the liver sometimes suppurate and give rise to abscesses.

II. CHRONIC INTERSTITIAL HEPATITIS OR CIRRHOSIS.

By these terms we signify a chronic inflammation of the interstitial tissue of the liver. It occurs in three forms.

I. DIFFUSE INTERSTITIAL HEPATITIS or FINE CIRRHOSIS.

This form of cirrhosis occurs as a result of malarial fever, and the organ may exhibit a melanæmic condition simultaneously. Other fevers occasionally cause a degree of cirrhosis.

Characters.—The liver is diminished in size and exhibits a finely granular surface. The tissue is tougher than natural. Microscopically, there is seen to be an increase of connective tissue between adjacent lobules in the region of the fine branches of the portal vein, compressing the peripheral cells and causing their atrophy.

II. COMMON CIRRHOSIS.

(Synonyms: "Coarse" Cirrhosis; "Hobnail Liver"; "Alcoholic" Cirrhosis ; "Gin-drinker's Liver.")

Causes.—(1) Alcohol, in the form of raw spirits especially. (2) syphilis.

Characters.—In a typical case of alcoholic cirrhosis, the liver is much smaller than natural, and very tough and firm. The surface is irregular, being studded with numbers of small flat projections resembling hobnails. The organ is anæmic, but the depressions around the hobnails are paler than the latter. Sometimes the surface has a general tawny yellow tint, due to bile staining. The capsule is thickened. On section, the liver tissue is seen to be divided into small areas by bands

Suppurating Hepatitis

1 Tropical abscess – solitary
2 Pyaemic Abscess – multiple
3 Cystic abscess
4 Suppurating Tumours

of dense fibrous tissue, each area containing a group of lobules and having a diameter of $\frac{1}{8}$ to $\frac{1}{4}$ of an inch, or thereabouts. The liver tissue is pale, unless bile stained.

Microscopically, the lobular structure is obscure. The central veins are compressed, perhaps obliterated, and the arrangement of capillaries is lost. The hepatic cells are closely packed together, and large numbers of them atrophy—partly from mechanical pressure and partly from deprivation of blood supply. The cells may show fatty changes. The bands of fibrous tissue are observed to be continuous with the deep layer of the capsule : the "hobnails" are simply groups of liver cells rendered prominent by the contraction of surrounding connective tissue. The connective tissue grows alongside of the medium sized portal veins, and the latter in consequence suffer compression. The tissue is at first entirely cellular, but as the disease advances it becomes more and more fibrous, contracting simultaneously and destroying the liver tissue. There is a considerable development of new blood-vessels in the fibrous tissue, and these can be injected from the hepatic artery, indicating their source of blood supply. The bile capillaries are comparatively unaffected, and consequently there is an absence of jaundice.

The hepatic veins being obstructed, the venous blood is carried off to a certain extent through a supplementary set of vessels in the capsule and suspensory ligament. The obstruction of the medium sized portal veins leads to a dropsical effusion from the larger veins of the portal system—ascites.

III. BILIARY CIRRHOSIS.

(SYNONYMS : "HYPERTROPHIC" CIRRHOSIS ; "UNILOBULAR" CIRRHOSIS).

This form of cirrhosis appears to result from obstruction in the bile capillaries. Charcot produced the condition artificially by ligaturing the bile ducts in an animal.

Characters.—These differ very markedly from the characters of common cirrhosis, so, for the sake of contrast, we shall adopt the same order of description here. The liver is increased in size, firm and brittle. The surface is smooth and deeply bile-stained ; and the capsule is finely granular like morocco leather, but is not thickened. On section, the tissue is seen to be extensively jaundiced ; the arrangement of fibrous tissue cannot be made out with the naked eye.

Microscopically, the connective tissue around individual lobules is increased in quantity, growing especially in relation to the bile ducts, which are thereby compressed. The periphery of the lobules is invaded to some extent by cellular tissue. Running in from the margin of the lobules are large numbers of bile capillaries, each one consisting of a double row of cells. The liver cells are considerably atrophied.

The portal veins are not involved, so there is no ascites. But the condition is accompanied by profound jaundice, due to obstruction of the bile ducts, and also by fever. A fatal result frequently follows, with symptoms like those of acute yellow atrophy.

SYPHILIS.

The liver may be affected with syphilis in congenital or acquired form.

I. SYPHILITIC CIRRHOSIS

Is found in the livers of children dying with the taint of congenital syphilis.

Characters.—The organ is enlarged, tough and firm, and heavier than usual. The tissue is either uniformly pale or exhibits pale and purplish patches merging one into the other. The pale patches are the sites of the most advanced changes, and are somewhat depressed: they have a pearly-white centre, surrounded by a yellow zone which merges into a congested zone further out.

Microscopically.—In the pale-coloured areas the lobules have suffered marked alteration. Around them and invading the liver cells, there is a great increase of fibro-cellular tissue, causing atrophy of the cells and compression of capillaries. This new tissue arises in a peculiar manner. It is derived from a proliferation of the endothelial lining of the capillaries and of the connective tissue corpuscles that line the lymph spaces between the capillaries and the liver cells.

The condition is often complicated with gummata, which are small and numerous.

II. GUMMATA

Are frequently found in the liver as a sequel of acquired syphilis. They commonly occur on the superior surface near the suspensory ligament, and are larger than those met with in the congenital form. They are, to begin with, firm and dry; as they become older, they caseate and become enveloped by a dense fibrous capsule, which contracts and forms a depression around them. The surrounding liver tissue is anæmic.

Microscopically.—In the early stage, the gumma consists of the tissue elements of the liver, infiltrated with numerous young cells. There is congestion around. The cells undergo various metamorphoses, evidenced by change of shape and loss of staining properties. The vessels become the seat of syphilitic endarteritis, and are gradually occluded. The occlusion may be precipitated by the formation of a thrombus. The gumma then caseates, and becomes gradually absorbed, leaving a cicatrix. A succession of these cicatrices imparts a lobulated appearance to the liver, due to contraction of the fibrous tissue.

TUBERCULOSIS.

This occurs in two forms in the liver.

Acute Tuberculosis leads to the formation of tubercle granulations in the connective tissue stroma of the liver and in the capsule. They are of very small size and soon caseate. The largest of them are generally bile-stained. They possess the typical structure of grey tubercles.

Chronic Tuberculosis of the liver occurs along with the same condition in the spleen and other organs. Larger tubercles form in this case; they appear to originate in the portal canals in the neighbourhood of the bile ducts. They frequently soften in the centre, and these softened areas have been described under the name of "bile cysts."

A **primary chronic tuberculosis** is occasionally observed in the liver. The disease seems to start in the bile ducts, and it extends along their walls, caseating and ulcerating into the lumen, giving rise to irregular cavities with rugged bile-stained walls.

LYMPHADENOMA,

Or "Hodgkin's disease," gives rise to large circumscribed growths in the liver, precisely homologous to the growths already described as occurring in lymphatic glands (*v.* page 94).

LEUCOCYTHÆMIA.

The liver, in common with other organs, is implicated in this disease.

Characters.—The liver shows considerable enlargement and increase in weight. The tissue is pale and very soft, and hæmorrhages are seen under the capsule as well as on section. The lobules are abnormally distinct, being marked out by faint white lines.

Microscopically.—There is seen to be an extensive leucocyte infiltration into the portal canals and fissures, and the intra-lobular capillaries are engorged with leucocytes. At points, minute hæmorrhages are evident, consisting mostly of leucocytes, and they destroy the liver cells in their neighbourhood. The engorgement of the capillaries leads to compression of the adjacent ~~air~~ cells and their subsequent atrophy. liver cells

CYSTS.

Various forms of cysts are found in the liver.

(1) **Simple cysts.**—These occur as single cysts, containing serum, and originate in dilatation of a bile duct as the result of obstruction by cancer or tubercle. At first they contain bile, naturally, but the bile acids, salts, and colouring matter are gradually absorbed, leaving a clear serous fluid.

(2) **Hydatid cysts.**—The cystic form of the parasite *tænia echino*

coccus frequently grows in the liver as a single hydatid cyst, situated in the right lobe of the organ. The structure of these cysts has been fully described in a previous connection (*v.* page 79). They often caseate and calcify. Sometimes they rupture and perforate the diaphragm. It is a point of clinical note that they tend to do this more frequently than tropical abscesses (*v.* page 215).

(3) **Cystic degeneration** of the liver is associated with the same condition in the kidney. How it arises is unknown, but it results in the formation of numerous small cysts with thin, fibrous, smooth walls, and containing a thin æqueous fluid.

(4) Occasionally one finds small cysts due to the parasite **pentastoma denticulatum** and **distoma hepaticum**.

TUMOURS.

Simple.—The commonest of these is the **cavernous angioma**, which grows at the surface, and appears as a purple patch about a third of an inch in diameter, slightly depressed below the level of the surface. Its microscopic characters have been before described (*v.* page 44). Two varieties of **adenoma** are found, both rare :—(1) A *reproduction of parts of the liver*, with typical liver structure, forming sharply defined solitary tumours, with larger lobules and greater vascularity than the normal tissue. (2) *True adenoma*, existing as pale soft masses, usually multiple, made up of liver cells and scanty capillaries, but devoid of bile ducts.

Malignant.—**Sarcomata** are almost invariably secondary. They may arise by direct extension from some neighbouring organ, when they form large soft masses ; or the primary tumour may be sitated in some distant organ, and in such a case the secondary growths are nodular and multiple. **Melanotic sarcoma** occasionally occurs as a secondary growth.

Cancer is the commonest and most important tumour in the liver. *Primary cancer* is of rare occurrence : it starts in the bile ducts, assuming an adenoid type. *Secondary cancers* are exceedingly common as a result of direct extension from the stomach and pancreas, also following cancer of the intestine, less common following cancer of the uterus and other distant parts. The growth occurs in the form of numbers of isolated tumours scattered through the organ, many of them projecting from the surface as pale rounded nodules, encircled by a hyperæmic zone. They are of encephaloid type at first, but tend to become scirrhous. A fibrous formation takes place within the tumour, leading to a cicatricial contraction and a dimpling or *umbilication* of the superficial nodules. At the same time they undergo fatty changes, and become yellow. Instead of a nodular growth, the tumour sometimes assumes a diffuse character, infiltrating the whole liver.

DISEASES OF THE GALL-BLADDER AND BILE DUCTS.

GALL-STONES.

By this we mean calculi made up of the constituents of the bile in varying proportions. They consist, in most cases, of aggregations of cholesterine, mucin and bile pigment, sometimes with lime salts.

Single Gall-stones are almost entirely composed of cholesterine. They exist in the form of semi-translucent, glistening, oval stones, soft and very light, with a granular surface. When cut across, they present a central nucleus from which the surrounding structure appears to radiate. Single stones are commonly found impacted in the neck of the gall-bladder.

Multiple Gall-stones are more often met with than the former. Sometimes two or three stones of large size are found ; at other times varying quantities of smaller ones, often reckoned by hundreds. They are always of light weight, and present different shades of colour, from pale yellow to brown, green and black. The surface invariably exhibits a number of facets : these are produced by the mutual pressure of the stones one upon another, and the more stones there are the more numerous, accordingly, are the facets. On section, they show a stratified structure, the strata being of dissimilar shades of colour.

Effects of Gall-stones.—The presence of gall-stones in the gall-bladder may lead to a catarrhal inflammation of the mucous membrane: in some instances the gall-bladder is found full of stones embedded in mucus. But the chief evil effects of gall-stones arise from impaction of a calculus in the neck of the bladder, the cystic duct or common bile duct. A frequent seat of impaction is the termination of the common bile duct. An obstruction at that point prevents the entrance of bile into the duodenum, and produces secondary results on the system of bile ducts, which will be presently mentioned. In addition, the gall-stone may ulcerate a passage into the duodenum, or, on the other hand, into the peritoneum, where it sets up inflammation.

OBSTRUCTION OF BILE DUCTS.

This may result from one of several causes, the most important being that just mentioned. The others may be noted briefly :—(1) Pressure of tumours ; (2) Parasites ; (3) Inflammation secondary to catarrh of the duodenum, affecting the distal end of the common bile duct. It should be kept in mind that the obstruction itself often leads to inflammation.

Effects.—These vary with the particular tube obstructed.

(1) Obstruction of the **cystic duct** tells on the gall bladder, which undergoes one of two changes. It sometimes atrophies, the secretion drying up and perhaps calcifying. But more commonly, the bladder

continues to secrete mucus, which has the effect of dilating the sac. Through time the latter becomes converted into a thin-walled cyst, filled with a clear watery fluid in which very little mucus can be seen. The bladder being unavailable as a storehouse for bile, its loss is somewhat compensated for by dilatation of the larger bile ducts.

(2) Obstruction of the **common bile duct** primarily leads to stagnation of bile behind the seat of obstruction. The gall bladder dilates to accommodate the ever-accumulating bile, and then the larger bile ducts along with the ductus choledochus itself, suffer distension. The lining membrane in all these situations becomes inflamed and frequently ulcerated, so that the wall of the bladder or any of the ducts may rupture, permitting the bile to escape. Within the liver, a process of absorption of the bile takes place from the capillaries, with the result that the hepatic cells are stained a deep yellow with the biliary colouring matter. Following this, the bile pigment is gradually deposited in the various tissues of the body, the skin acquiring the same yellow colour which is presented by the liver. These are the phenomena of *jaundice.* But in addition, the bile capillaries may, in course of time, dilate and form cysts ; these generally present ulcerated walls, indicating that inflammation probably predisposes to the dilatation. The hepatic cells become atrophied from pressure, an irritative overgrowth of connective tissue is set up, and in this way "biliary cirrhosis" results.

TUMOURS.

A simple **adenoma** may affect any part of the wall of the gall-bladder : usually it grows in the fundus and projects into the interior. Microscopically, it is seen to consist of a number of small spaces lined with columnar epithelium and containing a slightly pigmented mucus fluid. **Cancer** occasionally starts in the gall bladder and involves the liver secondarily. It is generally of encephaloid type.

DISEASES OF THE PANCREAS.

ATROPHY

Of the pancreas is observed to occur as a senile change along with wasting of other glandular organs, also in the course of debilitating diseases. The organ has been found atrophied in many cases of diabetes mellitus, and that has been attributed to some lesion in the solar plexus.

FATTY INFILTRATION

Of the pancreas is a very common occurrence, and is generally a sequel to senile atrophy. It also takes place in conditions of general adiposity. The gland preserves its shape : on section it exhibits a wasting of the glandular structure, which is largely replaced by fat.

HÆMORRHAGE.

Small multiple hæmorrhages are met with in the pancreas under similar conditions to those which cause them in many other organs. Some instances are on record of diffuse infiltration of blood into the gland, followed by sudden death. These cases are to be explained by pressure exerted by the tumid gland on the solar plexus, inducing reflex paralysis of the heart.

PANCREATITIS,

Or inflammation of the pancreas, exhibits different types. It was pointed out that a perforating ulcer of the stomach often sets up inflammation in the gland. This may result in suppuration. Interstitial inflammations are sometimes observed, the pancreas being indurated and showing loss of gland structure.

CANCER.

This is much the most important disease met with in the pancreas. Primary cancer nearly always grows in the head of the gland, and is generally of the **scirrhous** variety. It leads to obstruction of the pancreatic duct, or even the common bile duct, besides setting up secondary growths in the duodenum, less frequently in the liver or peritoneum. On the other hand, the pancreas is very liable to be affected with cancer originating in the stomach.

OBSTRUCTION OF PANCREATIC DUCT.

It has just been mentioned that this may result from cancer in the head of the pancreas. It also occurs, sometimes, by the formation of calculi : these are composed mainly of lime salts. Obstruction of the duct leads naturally to its dilatation by the pressure of the gland's secretion within the duct. The dilated duct may come to form a cyst as large as the spleen. Sometimes the duct dilates at a number of points, so that a series of cysts is formed, imparting a beaded appearance to the duct.

DISEASES OF THE URINARY SYSTEM.

1. *The KIDNEY.*

MALPOSITIONS

Of the kidney are not uncommon. The displacement may be congenital or acquired, and it takes place either in a vertical or transverse direction. The kidney has been found actually in the pelvis ("pelvic kidney.")

The term " floating kidney " is employed to indicate an undue mobility of the organ. As a rule one kidney only (generally the right) is concerned. The condition occurs mostly in multiparous women, and generally depends on an abnormal degree of laxity of the covering peritoneum. Sometimes it is associated with the presence of a meso-nephros.

MALFORMATIONS.

One kidney is sometimes absent or imperfectly developed. Total absence of one kidney is rare ; more commonly there is an arrest of development in the organ, which persists throughout life as a rudimentary structure.

In some cases, the two kidneys have become fused to form a single large kidney giving off two ureters. "Horse shoe" kidney is a modification of this condition ; it is formed by the union of two kidneys by a band of tissue stretching across the middle line. The band may be mere connective tissue or exhibit an actual renal structure, and it invariably exists at the lower part of the organ. Both ureters always pass in front of it.

These malformations are frequently associated with abnormalities in the vascular supply. Further, where one kidney is absent, the corresponding testicle and vas deferens in the male (or ovary in the female) may be absent.

Sometimes the fœtal lobulation of the kidney persists throughout life.

Cystic kidney.—This is a phenomenon of congenital origin, and is found both in the fœtus and in the adult. Both kidneys are always affected. Each organ is much enlarged and irregular on the surface. On section it is seen to be composed of a mass of variously sized cysts, with a small amount of intervening normal renal structure. The cysts are lined by a layer of flattened epithelium, and they are filled with a clear watery fluid, which is occasionally blood-stained. The fluid contains albumen, cholesterine and blood pigment, but none of the ordinary urinary constituents. As to the **origin** of the condition, it is due to the mixture of the meso-nephros with the meta-nephros, at an early period of intra-uterine life.

HYDRONEPHROSIS

Is the result of mechanical obstruction in some part of the urinary tract, whereby the urine secreted by the kidney is prevented from escaping into the bladder, and the pressure of accumulating urine exerts an injurious effect on the kidney. Such obstruction usually occurs in the ureter, either from the impaction of a calculus or the pressure of a tumour without. Less commonly, a stricture of the urethra is the source of obstruction.

Characters.—The urine, being continually secreted, gradually accumulates above the seat of obstruction, and the pressure tells on

the kidney. The result is distension of the pelvis and calices of the organ, with subsequent atrophy of the renal tissue. The papillæ disappear, and the calices gradually increase in size until the organ is metamorphosed into a series of sacs connected by septa of fibrous tissue. The cortex is represented by a thin fibrous layer bounding the sacs. The organ becomes very much enlarged, and presents a lobulated appearance owing to the bulging of individual calices. The capsule is considerably thickened. The contents of the sac consist at first of normal urine, but latterly the urinary constituents of the liquid disappear, and there remains simply a watery albuminous fluid.

ATROPHY

Of the kidney is a condition of little importance. It is sometimes caused by the mechanical pressure of a growing calculus impacted in the pelvis of the organ. A cirrhotic degeneration of the kidney leading to atrophy occurs in old age coincidently with atrophy of other organs. In young persons, atrophy of the kidney has been known to follow obstruction of the renal artery.

HYPERTROPHY

Of one kidney is supposed to occur in a compensatory manner when the other is absent or destroyed. It is manifested by an increase of renal tissue, with an augmented vascular supply.

CLOUDY SWELLING

Of the kidney occurs under similar conditions to those governing its occurrence in other organs.

Characters.—The whole organ is enlarged, and both on the surface and on section is paler than normal. The capsule strips off readily with the fingers. On section, both layers of the cortex are found to be swollen, and present a pale mottled appearance, the mottled parts being due to hyperæmia. The hyperæmia is best marked in the Malpighian bodies, in the superficial stellate veins, and veins at the bases of the pyramids.

Microscopically.—The most marked changes are found in the epithelium lining the convoluted tubules and tho ascending limbs of the looped tubules of Henle (*i.e.*, the most highly organised cells). The cells are swollen and project into the lumen of the tube irregularly, giving it on section a somewhat stellate appearance. The cells are more granular and more opaque than normal, the nucleus being frequently obscured. Some of the cells are in process of breaking down, and hyaline and granular casts are sometimes found. The condition frequently goes on to fatty degeneration.

FATTY DEGENERATION

Of the kidney has various causes. It is sometimes a sequel of cloudy swelling or inflammation; sometimes it is a result of general malnutrition—as in phthisis, anæmia, cancer; in other cases it is due to poisoning, *e.g.*, by phosphorus or alcohol, or in pyæmia or septicæmia. It also occurs in diabetes; it is frequently a complication of waxy disease; and plays an important part in catarrhal nephritis.

Characters of simple Fatty Degeneration.—In advanced stages the kidney is usually slightly smaller and paler than normal. The capsule strips readily. On section the organ is flabby, the cortex usually normal in thickness, and of a yellow colour—either uniform or mottled. The medulla is normal in appearance. The Malpighian bodies often stand out prominently, and the vessels at the bases of the pyramids are congested.

Microscopically.—In fatty degeneration, as in cloudy swelling, the epithelium of the convoluted tubules and ascending limbs of the looped tubules of Henle is the structure most affected. These cells are swollen, and contain numerous globules of fat, especially towards the bases of the cells. The swollen epithelial cells in some cases almost occlude the lumen of the tubules, and the cells are often granular and in process of disintegration. The collecting tubules are rarely affected, and the blood-vessels still more rarely, except in phosphorus poisoning.

WAXY DEGENERATION

Of the kidney is very common, and occurs in a large proportion of cases where there is waxy disease in other organs, especially when caused by syphilis. It is frequently superadded to other changes in the kidney of an inflammatory or degenerative nature.

The distribution of the disease follows the course of the vessels in a very definite manner. It usually first affects that part of the arterioles nearest to the point where they divide into capillaries; so in the cortex it affects the afferent arterioles, and in the medulla the straight arterioles. From the afferent arterioles it spreads into the capillaries of the glomeruli, and thence into the efferent arterioles and the capillaries into which they break up, and also backwards into the smaller branches of the interlobular arteries; thence it spreads successively along the larger vessels till the large interlobular arteries and veins become involved. In the medulla the degeneration runs a similar course; spreading from the arteriolæ rectæ, it involves successively the capillaries and veins between the straight tubules (Greenfield). In later stages the basement membrane of the tubules becomes affected, especially the basement membrane of the collecting tubes. It is very rare for the epithelial cells to be affected. As the disease progresses some fatty degeneration and ~~interstitial~~ inflammation may be superadded. al

Naked eye Characters.—In the early stage the kidney is slightly enlarged, its capsule strips readily, the surface is smooth, pale, and

glistening, and the venæ stellatæ unusually distinct. On section, the Malpighian bodies stand out prominently in the cortex as rows of glistening spots on a pale, mottled background ; there is usually some congestion at the bases of the pyramids, the rest of the section being pale. The addition of iodine maps out very distinctly the parts affected with the waxy change, even when only present to a slight degree. When the condition is more advanced, the kidney is usually much enlarged and its specific gravity increased ; capsule normal and surface pale as before. On section, the cortex, especially in its superficial layer, is much increased in thickness, and has a pale translucent appearance, with the Malpighian bodies standing out as large glistening masses. The striation at the base of the pyramids is very marked, and there is still some congestion in that situation.

Microscopically, the Malpighian bodies and the walls of those vessels affected with the waxy change have a swollen, translucent appearance ; the degeneration is seen to pick out the middle coat of the arterioles ; owing to the thickening of the walls of the vessels their lumen is diminished. There are fatty changes in the epithelium lining the tubules, though it is very rare for these cells to be attacked by the waxy degeneration. Numerous colloid casts are to be seen filling the tubules, and causing flattening of the epithelium surrounding them ; such casts do not take on the ordinary staining reactions of waxy material : granular casts are sometimes also present. In advanced cases inflammatory changes (most frequently catarrh, especially of collecting tubes, rarely interstitial inflammation) are apt to be superadded.

There is a rare form of waxy degeneration, in which the changes are limited to the medulla and do not follow the typical regular order. The change commences at the apex of the papilla and affects chiefly the connective tissue fibres between the straight tubules, and to a less extent the walls of the vessels there. The papillæ have a translucent appearance. Along with this there is often a catarrhal process in the collecting tubes, and sometimes it is associated with waxy degeneration of the pelvis of the kidney.

Combined forms of waxy disease with other changes in the various structures of the kidney are extremely common.

(1) A combined **waxy and fatty** kidney is commonly found in wasting diseases, *e.g.*, phthisis. The appearance of the kidney, which is greatly enlarged, is similar to that described under waxy kidney, with, in addition, well-marked yellow mottling in the region of the convoluted tubules.

(2) A very frequent combination is that of **waxy kidney with catarrhal changes** of a chronic nature in the convoluted tubules, unattended by fibroid thickening of the vessel walls. The kidney is greatly enlarged, capsule strips normally, and surface is smooth. On

section the organ presents a pale mottled appearance, but the chief feature is great swelling of the cortex in both its superficial and deep layers.

(3) **Waxy and interstitial** changes also occur together in the kidney, this is most common in syphilis and phthisis (Greenfield). This condition may be attended either by enlargement or by contraction of the kidney, according as the increased interstitial growth of connective tissue is associated with catarrh or atrophy of the epithelium lining the tubules. In the *first* variety, where the kidney is enlarged, the capsule is thickened and somewhat adherent, and on stripping it off the surface is granular and somewhat pale in colour. On section the superficial cortex is observed to be thinned, while the inter-pyramidal cortex is much thickened. There is a mottled pallor, with some translucency—especially in the superficial cortex. Some cysts are usually present.

The *second* variety of combined waxy and interstitial change is a more advanced condition, and has been described as the third or atrophic stage of waxy kidney. The organ is very small; capsule is adherent at parts, and, on stripping it off, an irregular granular surface is left. The whole section has a pale translucent appearance. The superficial cortex is very narrow and sometimes contains minute cysts, while the inter-pyramidal cortex may be normal. Microscopically, the vessels are found extensively affected with waxy disease, and there is considerable interstitial inflammation. This condition seems to be invariably due to syphilis.

HYALINE DEGENERATION

Of the kidney occurs in scarlet fever. It affects chiefly the afferent arterioles and glomerular capillaries, and also to a slight extent the interstitial connective tissue.

CALCIFICATION

Of the kidney occurs sometimes without obvious cause, in other cases it has been ascribed to metastasis due to extensive absorption of lime salts from the bones, *e.g.*, in osteomalacia and rickets. Simple calcification affects chiefly the walls of the arteries; occasionally, when the kidney has become functionally useless and has undergone fatty degeneration, *e.g.*, after obstruction of the renal artery, the whole organ calcifies. Calcareous nodules may also be found in old caseous infarcts.

VENOUS CONGESTION

Of the kidney is observed in the bodies of those who have died slowly from any cause, and also in cases of death preceded by coma. The congestion is most marked in the following situations—in the venæ stellatæ, at the bases of the pyramids, and in the Malpighian bodies.

CHRONIC VENOUS CONGESTION.

Causes.—Chronic valvular disease of the heart, especially mitral disease; chronic lung disease, *e.g.*, phthisis, chronic bronchitis, &c., venous obstruction—from thrombosis of inferior vena cava or more rarely of renal veins.

Characters.—The kidney is enlarged, capsule strips normally, surface smooth, venæ stellatæ engorged. On section, the surface is very dark in colour, the organ feels firm and elastic, and exhibits sharp edges. The cortex is somewhat thickened, the Malpighian bodies stand out prominently; there is marked congestion at the bases of the pyramids, and the engorged venæ rectæ render the striation of the medulla very distinct. When the condition is due to heart disease, infarcts are frequently present in the same kidney.

Microscopically, all the veins are seen to be distended, their walls thickened, and some fibrous induration around them. The glomerular capillaries are also much distended and their walls markedly thickened: the intertubular capillaries share in the general engorgement. Frequently extravasated blood is found in the convoluted tubules, owing to rupture of some of the glomerular capillaries; farther on in the course of the urinary tubules, this blood has undergone changes, the blood corpuscles breaking down and the protoplasm becoming absorbed, the blood pigment being left in the form of casts—often quite black, which are called "melanin" casts. Frequently also there is catarrh, especially in the collecting tubules, leading to the formation of casts. In advanced cases there is sometimes atrophy of the epithelium of the convoluted tubules.

OBSTRUCTION OF ARTERIES.

I. OBSTRUCTION OF THE RENAL ARTERY

Is not very common. The most common cause is atheroma—leading to thrombosis, which usually spreads from the aorta into the renal artery. Sometimes the obstruction is due to embolism.

Results.—The entire kidney undergoes fatty degeneration, becomes adherent to the surrounding structures owing to inflammation, atrophies, and ultimately calcifies.

II. OBSTRUCTION OF BRANCHES OF RENAL ARTERY

Is usually due to embolism, the embolus coming either from the heart or from an atheromatous patch in the aorta. The result is infarction. Infarcts are often multiple and of various sizes, and frequently associated with chronic venous congestion. The infarcts run the same course as in other organs. In the early stage they are sometimes pale and colourless, not raised above the surface, and surrounded by a hyperæmic zone; in other cases they form a dark red, raised, wedge-shaped patch in the cortex, hæmorrhage having occurred; the red colour is most marked at the periphery, the centre being usually pale and yellowish. Later on, a process of fatty degeneration and caseation occurs, and calcification may follow; rarely softening occurs, and small cysts are formed in the cortex. Usually the infarct becomes gradually absorbed, and its place is taken by dense fibrous tissue which forms a depressed cicatrix on the surface of the kidney, over which the capsule is adherent.

III. OBSTRUCTION OF SMALLER ARTERIAL BRANCHES—CAPILLARY EMBOLISM.

Causes.—**Bacteria** in various diseases, *e.g.*, ulcerative endocarditis, pyæmia, anthrax, diphtheria, tuberculosis, &c. The bacteria form minute plugs in the afferent arterioles and capillaries of the glomeruli. In many cases this capillary embolism seems to cause no disturbance; when very extensive, the capillaries become distended, some of them rupture, and the organisms pass into the urinary tubules, where, if they remain, they lead to inflammatory changes.

Fat embolism occurs in some cases of diabetic coma, sometimes, also, after fractures. The fat in the blood becomes entangled in the glomerular capillaries, and the effects are similar to those just mentioned. The kidney is somewhat enlarged and pale, with minute hæmorrhages towards the surface. Microscopically, fat globules can be detected in the capillaries; around these points there is some congestion, with small hæmorrhages; and fat globules may be found in the neighbouring tubules.

TUBE CASTS IN RENAL DISEASE.

A tube cast consists of an aggregation of solid or partially solid material, which collects within the renal tubules and becomes moulded on their walls, so as to form a cast of their interior.

Varieties.—(1) Casts composed of altered blood or of some exudation (fibrinous or albuminous) from the blood; (2) Casts arising from some of the tubular structures—detached or broken down epithelial cells or leucocytes; (3) Casts formed from substances which are being excreted through the urine and become crystallized or consolidated in their passage through the tubules, *e.g.*, bile pigment, uric acid, or sodium urate (Greenfield). Combinations of these varieties often occur.

(1) CASTS DERIVED FROM THE BLOOD.

Blood casts are found in cases of acute congestion or acute inflammation of the kidney, and in chronic venous congestion of the kidney. In the acute cases the casts are composed of almost unaltered blood corpuscles, and *post-mortem* are found chiefly in the convoluted tubules. In the more chronic cases, the casts are found throughout the course of the tubules in various stages of transformation: in the proximal part of the tubules there are found casts composed of blood corpuscles; lower down the corpuscles have disintegrated, and the casts are com-

posed of crystalline or granular blood pigment of a golden-brown colour (haematin or hæmatoidin casts); still farther on the casts are black (melanin casts).

Hyaline casts are composed of fibrinous exudation with some mucin, and are almost completely translucent. They are found chiefly in the looped tubules of Henle and in the larger collecting tubules. They are sometimes found in healthy urine, but chiefly occur in cases of acute inflammation. Epithelial cells, granules, &c., are often accumulated round a central hyaline cast.

(2) CASTS DERIVED FROM ALTERED TUBULAR STRUCTURES.

Casts are frequently composed of **epithelial** cells in various stages of disintegration, resulting either from a catarrhal process with detachment of the epithelial cells, or from granular or fatty degeneration of the cells. The cells are sometimes unchanged or somewhat fatty, and frequently surround a central hyaline cast: such casts are found in cases of acute catarrh. **Granular casts** consist of granular matter round a hyaline centre. In **fatty casts** the epithelial cells have broken down into oil globules. **Colloid casts** consist of homogeneous yellowish material, not so translucent as hyaline casts, and often showing transverse cracks. They occur in cases of waxy degeneration, also in the various forms of renal inflammation. They are probably formed by the coalescence of altered epithelial cells.

(3) CASTS FORMED FROM SUBSTANCES EXCRETED BY THE KIDNEY.

In cases of long continued jaundice there are found dark granular casts containing **bile pigment.** Crystals of **uric acid** are sometimes found in the kidney, especially in infarcts. In cases of granular contracted kidney yellowish deposits of **acid urate of soda** are found in the tubules. Casts of **calcium carbonate** occur in cases of osteomalacia. Sometimes epithelial casts undergo calcification, especially in the collecting tubules.

NEPHRITIS.

This signifies inflammation of the kidney, a condition which is the most prominent feature in "Bright's disease." The kidney may be looked upon as composed of three main groups of structures:—(1) Vascular structures; (2) Connective tissue and lymphatics; and (3) Tubules and the epithelium lining them. The changes in each of these structures manifested in nephritis will now be considered in detail.

I. Changes produced in the vascular structure by inflammation.—All the vessels of the kidney become engorged: the hyperæmia is specially marked in the Malpighian bodies and adjacent capillaries; frequently some of the glomerular capillaries rupture, and blood escapes into the tubules. Along with the hyperæmia, there occurs transudation of lymph from the capillaries into the tubules (causing the production of hyaline casts and albuminous urine) and in the connective tissue between the tubules. Leucocyte emigration also takes place, and in the cortex is most marked in the vicinity of the glomeruli and along the interlobular vessels. Only in extreme cases is it well marked between the tubules. In the medulla the emigration occurs around the bundles of straight vessels. Hyaline degeneration attacks the vessel-walls, especially the afferent arterioles, sometimes also the glomerular capillaries (well seen in fevers). Along with this there is often marked connective tissue proliferation in the middle coat. As the inflammation becomes chronic, both periarteritis and endarteritis occur; a diffuse fibrous induration surrounds the vessels and spreads into the neighbouring structures. The epithelial cells of the glomerular capillaries and the connective tissue cells between the capillaries become swollen, proliferate, and sometimes lead to obstruction of the capillary tuft. Frequently, also, the basement membrane of Bowman's capsule swells, and the epithelial cells lining the capsule and on the tuft proliferate and form masses of cells, between which connective tissue fibres develop. The capillary tuft becomes adherent to the capsule, and ultimately in extreme cases the glomerulus becomes obliterated by fibrous tissue. When the condition has reached this stage there is great thickening of Bowman's capsule, and much fibrous induration around it.

II. **Interstitial changes.**—Lymph exudation and a certain amount of leucocyte emigration occur around the vessels into the spaces between the tubules. At an early stage, proliferation of the intertubular connective tissue commences, and this in time tends to produce degeneration and atrophy of the tubules. Another theory is that the interstitial changes are the sequel of catarrhal changes in the tubules.

III. **Changes in the Tubules.**– These are specially prominent in the most highly organized parts of the tubules, viz.,—in the epithelium of the convoluted tubules, and of the looped tubules of Henle. In the convoluted tubules there first occurs the process of cloudy swelling, followed by proliferation, granular degeneration, and detachment of the cells, which form masses blocking up the tubules. The place of the original cells is taken by new cells of irregular form and size, which in their turn are constantly being cast off and replaced. The detached epithelium forms casts, which are carried along the course of the tubules and become discharged in the urine. The cells frequently undergo rapid fatty or granular degeneration (fatty and granular casts); in other cases they undergo colloid degeneration, and become fused into homogeneous masses (colloid casts); some of the disintegrated catarrhal cells become absorbed.

Later on, fresh changes occur in the convoluted tubules: either (1) the tubules atrophy, or (2) repair takes place—the lining of the tubules being formed by small flattened cells which have developed from the

original epithelium. In chronic cases, these changes are accompanied by interstitial nephritis, and sometimes by swelling of the basement membrane. In catarrh of the looped tubules of Henle the changes are almost identical. In catarrh of the collecting tubules the epithelial cells which develop are smaller, and there is a less degree of detachment of the cells.

Inflammation may attack the different structures of the kidney in different degrees: when the glomeruli and vessels are chiefly affected, the process has been called "glomerulo-nephritis"; where the tubules are chiefly affected it is called "catarrhal, parenchymatous, or desquamative nephritis"; where the intertubular tissues are most affected it is called "acute or subacute interstitial nephritis."

I. ACUTE NEPHRITIS.

Cause.—Generally some irritant conveyed by the blood; certain fevers and blood diseases, *e.g.*, scarlatina, septicæmia, &c.; poisoning by cantharides, turpentine, etc.

Scarlet Fever is one of the commonest causes of acute nephritis, especially in children, and it produces microscopic changes in the kidneys exactly corresponding to those just described. In the acute stage the kidney is enlarged (especially at the cortex), very soft, and intensely congested; frequently hæmorrhages are to be seen, especially beneath the capsule and at the bases of the pyramids. *Microscopically*, the changes are those of acute nephritis, with usually a marked degree of hyaline degeneration in the vessel walls. In the majority of cases the inflammation subsides, and leads to no farther results. Sometimes, however, the inflammation advances and develops into **Post-Scarlatinal Nephritis**, which usually sets in during convalescence from the fever. The kidney is now only slightly enlarged; of firm consistence; capsule normal; suface smooth, pale in colour, and more translucent than normal. On section, the superficial cortex is slightly atrophied and mottled with dark points corresponding in position to the enlarged glomeruli. The interpyramidal cortex is greatly swollen, pale, with yellow mottling, and dark spots scattered through it as in the superficial cortex. *Microscopically* the chief changes are found in and around the glomeruli, and correspond to those already described; briefly they are as follows,—leucocyte emigration around the vessels, and extending round the glomeruli; proliferation of cells in the glomerulus and lining Bowman's capsule; swelling of the capsule; changes in the tubules only to a slight degree, as a rule, but in other cases there is intense catarrh; similarly there is a variable amount of interstitial change. The condition may ultimately resemble that of the granular contracted kidney.

There is a rarer form of nephritis occurring in scarlet fever complicated with *pyæmia*, in which the kidney (especially the cortex) becomes greatly swollen, pale in colour, and very soft. Microscopically, glomerular changes are found in an advanced degree, along with intense inflammation in the other structures.

In **ordinary acute nephritis** the kidney is enlarged, the capsule tense and strips very readily. The surface is pale and mottled with prominent stellate veins. On section, the cortex is swollen in both its layers and has usually a pale mottled appearance, but the congested glomeruli and vessels stand out as pink points and lines. Sometimes minute ecchymoses are seen in the cortex. The microscopic changes have been already described.

II. SUBACUTE NEPHRITIS

May be primary or may follow an acute attack. The most marked changes are, as a rule, in the **interstitial** tissues.

Naked eye Characters.—The organ is enlarged and of firm consistence; capsule normal. On section, the surface presents a pale mottled appearance, with yellowish streaks due to fatty degeneration. The superficial cortex is somewhat atrophied, while the deep cortex is swollen. The Malpighian bodies are not prominent. Occasionally a few small cysts are to be found.

Microscopically, there is found increased interstitial connective tissue formation, which occurs principally in the regions of the interlobular arteries and glomeruli in the form of dense patches of connective tissue (often very cellular), causing separation of the tubules from each other; between these patches the kidney tissue is comparatively normal and open. Many of the glomeruli themselves undergo fibrous degeneration and atrophy; while those in the more open tissue often appear perfectly normal. The tubules also suffer, and either present the appearances of chronic catarrh or have undergone atrophy. Increased connective tissue growth also occurs in and around the vessel walls, both periarteritis and endarteritis being present in advanced cases. Ultimately these changes lead to a condition closely resembling the granular contracted kidney.

III. CHRONIC INTERSTITIAL NEPHRITIS.

(SYNONYMS: GRANULAR CONTRACTED KIDNEY, CIRRHOTIC KIDNEY, GOUTY KIDNEY.)

This may be a sequel of acute or sub-acute nephritis. In some cases it results from a slow process of degeneration accompanied by arterial sclerosis throughout the body; this may be simply a senile change, or may be associated with gout, lead poisoning or chronic alcoholism. The onset of the disease is insidious and its course extremely slow.

Naked eye Characters.—The kidney is greatly reduced in size. The capsule is thickened and firmly adherent to the kidney and also to

the surrounding fat. On attempting to strip off the capsule, it separates in layers, and cannot be completely removed without bringing with it portions of the cortex. The surface of the kidney is extremely granular, and feels like morocco leather, being studded over with minute projections about the size of a pin head, each of which is pale in the centre and is surrounded by a redder injected furrow. In the cortex are numerous cysts, varying in size from very minute cavities to others as large as a walnut; these cysts contain a serous, seldom bloody, fluid. Reddish points are also scattered over the cortex, these being crystals of uric acid or sodium urate. The superficial cortex is extremely narrowed; in the earlier stages the inter-pyramidal cortex is often swollen, in advanced cases it becomes atrophied. The normal regular striation of the cortex is either altogether obliterated or has become tortuous and irregular; the inter-lobular arteries are also tortuous and thickened. The colour of the organ varies; usually it is dark red, but occasionally it is very pale. There is often an abnormally large amount of fat in the hilus of the kidney.

Microscopical appearance.—The changes are most marked towards the surface of the cortex. Passing inwards from the depressions on the surface of the cortex, there are wedge-shaped masses of dense fibrous tissue—the base of the wedge towards the surface; these masses correspond to the lines of the interlobular vessels. Between the masses there are more open patches in which the tubules are either normal in size or dilated. The denser bands are composed of fibrous tissue, in the centre of which is the thickened and tortuous inter-lobular artery: the Malpighian bodies have lost their regular arrangement and are grouped together more closely than normal; they show various stages of degeneration and atrophy. In the dense bands are also seen sections of atrophied convoluted tubules lined with flattened cells. The arteries show great thickening in all their coats.

As regards the Malpighian bodies, their capsule is thickened and laminated, and is adherent to the tuft of capillaries, the latter being sometimes atrophied, sometimes still patent; the afferent arteriole is much thickened; the thickening may spread to the basement membrane of the tubule, so as to obstruct the outlet of the glomerulus, thus leading to the formation of a cyst. **Cysts** in granular contracted kidney may be produced (1) by dilatation of glormeruli as above; (2) by dilatation of tubules, caused by their constriction owing to twisting or formation of casts, especially colloid casts. A considerable length of tubule may be dilated into a cyst, or a series af small cysts may be formed in a tubule. The cysts vary in size; the small ones contain colloid material, the larger ones contain a mucoid or bloody fluid; the epithelium lining the cysts is flattened and atrophied.

Granular contracted kidney is associated with hypertrophy of the left ventricle of the heart, thickening of vessels throughout the body, and frequently with cerebral hæmorrhage.

IV. SUPPURATIVE NEPHRITIS.

Suppuration in the kidney is most commonly disseminated, but sometimes there are localized abscesses (*e.g.*, suppuration of cyst, or in pyæmia).

Causes of suppurative nephritis.—(1) Carriage of masses of fibrin, etc., containing bacteria into the vessels of the kidney, and their lodgment there giving rise to intense inflammation, *e.g.*, in pyæmia, ulcerative endocarditis, etc. (2) Irritation by septic urine, *e.g.*, in cases of cystitis. This gives rise to "surgical kidney" or suppurative pyelo-nephritis.

In *pyæmia*, abscesses of the kidney are not common; they may be multiple or solitary, and affect one or both kidneys. They are chiefly situated in the cortex, and form wedge shaped masses near the surface.

Abscesses in the kidney occurring in the course of *ulcerative endocarditis* are usually in the form of numerous minute wedge shaped masses in the cortex, surrounded by a zone of intense injection. The whole organ is much enlarged and softened.

Surgical Kidney,—"Suppurative pyelo-nephritis" or "disseminated suppurative nephritis." In this condition both kidneys are, as a rule, affected, one usually more so than the other. It is commonly associated with disease of the bladder and of the pelvis of the kidney. The pelvis is frequently dilated and acutely inflamed. The inflammation may be limited to the calices, or may be more widespread: its mucous membrane is congested, and covered with a layer of muco-pus and earthy phosphates. The urine is often ammoniacal. In the early stage the kidney is swollen and congested; capsule strips readily. On the surface are yellowish points, either scattered or in small groups, projecting above the surface and surrounded by a zone of injection: the rest of the surface appears normal. As the case progresses, these yellow spots increase in number. On section the projections are found to correspond with small linear areas of suppuration in the cortex, which are surrounded by intense injection. Similar areas are found throughout the cortex and medulla.

Microscopically, there is found intense inflammation, with abundant leucocyte emigration around the interlobular arteries and glomeruli. The epithelium of the tubules is in a condition of cloudy swelling. As the process advances, the whole of the tissues become infiltrated with leucocytes, necrosis occurs, and small abscesses are formed. In the medulla similar changes occur. Numerous bacteria are found in the abscesses, as well as in the tubules and vessels. In more advanced stages the abscesses are larger and ragged; the kidney is often one mass of abscesses, and some of these communicate with the pelvis of the

kidney. The condition is almost invariably fatal, especially when bilateral. If unilateral, cure sometimes results, the kidney becomes contracted and adherent to surrounding structures, and the abscesses dry up and form caseous masses.

Pathology.—Most probably it is due to the entrance of bacteria into the kidney, probably through the vessels. Formerly it was held that the bacteria travelled along the tubules to the cortex, but this is doubtful, as the inflammation chiefly occurs round the vessels. Other theories as to causation :—(1) Rupture of over-distended glomeruli and escape of urine into the surrounding tissues ; (2) Absorption of putrid urine from the pelvis ; (3) Ordinary pyæmic abscesses ; (4) Reflex irritation from neck of bladder.

Pyonephrosis, or abscess in the pelvis of the kidney. This is due to irritation in the pelvis, sometimes caused by calculi (especially oxalic). Inflammation is set up, sometimes attended by hæmorrhage, and followed by ulceration and suppuration. The calculus may block the ureter completely ; sometimes it passes entirely or in part into the bladder. Another cause of pyonephrosis is over distension of the pelvis, brought about in the same way as hydronephrosis. The pus does not escape by the ureter, but burrows through the surrounding tissues in various directions.

Perinephritic abscesses have usually no organic connection with the kidney. Sometimes they are caused by the extension of suppuration from the kidney, and in some cases appear to be due to the rupture of a cyst on the surface of the kidney.

TUBERCULOSIS.

In general acute tuberculosis, the kidney becomes the seat of **miliary tubercles.** These are distributed in considerable numbers throughout the cortex of the organ, a few being found in the medulla. In a section of the kidney they are seen to be disposed in groups, and frequently exhibit a linear arrangement radiating from the periphery inwards. They are grey and translucent, soft and prominent above the surface of section ; and they are very liable to early caseation. The growth of the granulations commences around the interlobular arteries.

Renal phthisis or **tubercular pyelo-nephritis** is a local manifestation of tuberculosis, involving the kidney and urinary tract as well as the internal organs of generation. The disease is commoner in males than females. In the former, it originates in the kidney or in the epididymis of the testicle, and spreads by direct continuity. As a rule only one kidney is involved, but the second one may become affected by direct extension from the other by way of the ureter and base of the bladder.

Morbid Anatomy.—In the *kidney,* the process starts either in the mucous membrane of the pelvis or calices, or in the papillæ, and develops the phenomena of tubercular ulceration. It originates in tubercle granulations, which grow in large numbers and ulcerate as soon as they are formed. As a result of this, the pelvis and calices become dilated into large pouches with ragged caseous walls, while the kidney substance becomes more or less riddled with small cavities (resembling phthisical cavities), having caseous ill-defined walls and soft putty-like material in their interior. In consequence of these changes, the kidney becomes enlarged and irregular in shape ; the capsule is firmly adherent to the surface, and the surrounding tissues are as firmly attached to the capsule. It is not uncommon for one of the cavities to open on the surface and cause an abscess in the lumbar region. The *ureter* is thickened and cord like. Its mucous membrane is especially involved, being infiltrated with the tubercles and extensively ulcerated. The tube becomes blocked with the caseous debris, and this is no doubt a factor in promoting the dilatation of the pelvis—the passage of urine being impeded. The *bladder* exhibits tubercular infiltration at its base, chiefly between the ureters. Ulceration may follow : the ulcers are quite superficial, and exhibit pale coloured edges.

SYPHILIS.

The kidney is rarely the seat of syphilitic lesions, but syphilis is an important cause of waxy disease in the kidney, and in particular that form associated with interstitial nephritis.

LEUCOCYTHÆMIA.

(*v.* page 89.)

The kidney frequently becomes affected in this disease. The organ in advanced cases exhibits a dull white colour, relieved by red spots due to capillary hæmorrhage. These hæmorrhages are often very numerous, and occur under the capsule as well as in the substance of the organ. Microscopically, one finds an enormous infiltration of leucocytes in the renal tissue, separating the tubules. Many of the vessels are actually obstructed with the cells, and this "leucocyte thrombosis" appears to give rise to the hæmorrhages. Notwithstanding these changes, the function of the organ may be carried on, though imperfectly.

TUMOURS.

(1). **Primary.**—Of simple tumours, **fibromata** are the commonest. They usually grow in the papillæ, where they form minute circular nodules, firm and translucent. They are apt to be mistaken for tubercles, but the latter rarely infest the papillæ, and careful examination enables one to recognize their distinctive characters. Of malignant tumours, **cancer** occasionally develops primarily in the kidney. It gene-

rally attacks only one kidney, forming a soft encephaloid tumour which may involve the whole or only part of the organ. It is very liable to cause erosion of blood-vessels, resulting in hæmaturia. Cancerous emboli are also apt to be carried to the lungs, setting up a secondary growth there. In the second place, **lympho-sarcoma** sometimes originates in the kidney. It is often mistaken for cancer in children.

(2). **Secondary.**—The kidney is not a common site for secondary growths. Cancer and sarcoma are both occasionally found.

PARASITES.

Various vegetable parasites are found in the kidney. In cases of anthrax, the **bacillus anthracis** is found in the capillaries of the glomeruli, also in the tubules. In the latter situation, the process of spore formation may be observed. The **actinomyces** fungus sometimes sets up secondary growths in the kidney.

Hydatids occasionally infest the kidney. One cyst only is formed, resembling that in the liver. The **filaria sanguinis hominis** is sometimes found in the vessels and lymphatics: it gives rise to chyluria. An organism which is found in tropical climates and now recognized as an exciting cause of hæmaturia is the **Bilharzia hæmatobium.** It inhabits the pelvis of the kidney.

RENAL CALCULI.

Calculi are concretions of the normal or abnormal solid constituents in the urine. They are usually formed in the pelvis of the kidney, although no doubt they very often originate in the tubules, and are discharged into the pelvis, where they increase in size by successive depositions of solid particles on their exterior. They may continue to grow within the pelvis, or be carried along the ureter to the bladder, and grow larger there. Very often small calculi are evacuated through the urethra during the act of micturition.

Under certain circumstances we find solid concretions in the *uriniferous tubules.* When children die within the first two weeks of their existence, an examination of the kidney very often reveals quantities of amorphous urates within the tubules, particularly towards the apices of the pyramids. The urates are mainly compounds of ammonia and uric acid, and they appear to the naked eye as yellow or reddish lines, converging to the apex of the pyramid. Lime salts are deposited in the same situation in certain bone diseases, a fact already referred to as an example of calcareous metastasis (*v.* page 5). The deposit imparts an appearance of white streaks along the course of the tubules. Again, in the case of newly born children dying of jaundice, the biliary colouring matter is found deposited in crystalline form in the tubules.

In the disease of gout, crystalline masses of urate of soda are deposited (among other situations) in the connective tissue of the kidney (*v.* page 10).

Calculi in the *pelvis* of the kidney are of varying composition, and generally contain more than one substance. The most frequent constituents are uric acid, phosphates, and oxalate of lime. The cause of the deposition of calculi is not always apparent, but it is certain that uric acid and oxalate of lime can only be precipitated from acid urine, and phosphates from alkaline urine. An excess of any of these substances in the urine favours their precipitation. **Uric acid** calculus is the commonest. It usually presents the appearance of a small round or oval stone with a finely granular surface, of hard consistence, yellow or red in colour. When these exist in considerable number, they constitute what is popularly known as "gravel." But gravel does not as a rule consist of pure uric acid : the latter is generally mixed with oxalate of lime, the two sometimes forming alternate layers. In some instances, uric acid crystals aggregate into large masses, which may completely fill up and distend the pelvis and calices. Such calculi cause gradual destruction of the kidney substance by their presence, and besides they are liable to get fractured, lacerate the kidney, and cause hæmorrhage into the bladder. **Urates** very rarely form calculi alone ; but they are frequently found in combination with uric acid. **Oxalate of lime** forms a component part of many calculi, including gravel stones. The calculus proper is a rounded ball, grey or dark brown in colour, and tuberculated on the surface like a mulberry. Hence it is generally spoken of as the "mulberry calculus." It is very hard and heavy for its size. The **phosphatic** calculus is very common. It nearly always consists of triple phosphate of ammonium and magnesium, rarely of calcic phosphate, and often has a nucleus of uric acid. "The phosphates are deposited from alkaline urine as a light, bulky, white substance, which is commonly very brittle." This calculus is more frequently found in the bladder than in the kidney. **Cystin** calculi are somewhat rare, occurring in individuals who are subjects of "cystinuria." That term signifies a condition in which crystals of cystin are present in the urine, at the expense of uric acid in some degree. Under these circumstances, calculi are prone to form in the pelvis of the kidney or in the bladder. They are oval in shape, yellowish in colour, with a crystalline granular surface, and possess the consistence of wax. The typical hexagonal plates or prisms may be detached from the surface, but sometimes the calculus has a coating of phosphates.

Effects of calculi on kidney.—(1) Atrophy of the kidney substance, and ulceration of the pelvis and calices, often leading to hæmaturia. (2) Pyelitis—inflammation of the pelvis. This may become suppurative. (3) Obstruction to the outflow of urine, and consequent hydronephrosis.

II. The BLADDER.

DILATATION.

Of the bladder results from (1) obstruction to the outflow of urine through the urethra, or (2) paralysis of the muscular coat, or (3) catarrh of the mucous membrane. The obstruction may be due to (*a*) enlargement of the prostate gland, (*b*) stricture of urethra, either of which will cause hypertrophy of the muscular coat in addition to dilatation. The presence of calculi or tumour growths may also induce hypertrophy of the bladder wall.

When the muscular coat is markedly hypertrophied, a magnified view is obtained of the interlacing bands of fibres, which are greatly thickened and very prominent beneath the mucous membrane, so that the interior of the bladder bears a resemblance to the internal surface of a ventricle of the heart. The depressions between the muscular bands come to form pouches, which often project beyond the serous coat of the bladder, so as to form diverticula. The urine stagnates in these sacs, and decomposes, with the result that inflammation is set up. This allows of still further distension. Such a diverticulum has been frequently found behind the bladder, in some instances being larger than the bladder itself. Deposits of phosphates take place in the sac, and calculi may form. Sometimes, the inflammation which is set up weakens the wall to such an extent that the wall of the sac ruptures, allowing of extravasation of urine into the peritoneum. The urine in the sac being septic, this terminates fatally by way of acute peritonitis.

MALFORMATIONS.

There are two malformations of the bladder which are worthy of note.

1. **Extroversion** of the bladder is a condition where the anterior abdominal wall is deficient above the pubis, the bladder being represented by its posterior wall which fills up the gap in the abdomen. The mucous surface of the bladder wall is thus exposed to the air, and the urine which trickles from the orifice of the ureters escapes over the pubis, there being no receptacle for it. This leads to excoriations in the skin, and besides the mucous membrane is in a state of chronic inflammation.

The extroversion is always associated with other defects of the generative organs.

2. **Patency of Urachus.**—The urachus—which is the persistent fibrous remnant of a portion of the allantois—occasionally does not close when its fœtal function is fulfilled, but continues to exist as a patent tube. This is generally the result of some obstruction in the urethra. In consequence of its patency, the urine escapes through the urachus, by way of the umbilicus. Sometimes the urachus dilates into a cyst, lying above the bladder.

CYSTITIS,

Or inflammation of the bladder, is nearly always due to the agency of bacteria, which produce decomposition changes in the urine and lead to irritation of the mucous membrane. In a large number of cases, bacteria are introduced into the bladder on a catheter, which has not been rendered aseptic. But in addition, there is generally some lack of vitality in the bladder wall which favours the growth and development of these organisms, for the passage of a dirty catheter does not by any means invariably set up cystitis. The virus of gonorrhœa may reach the mucous membrane of the bladder and give rise to inflammation, and the presence of a calculus tends to have a similar effect.

Characters.—The inflammation is of a catarrhal nature, and may be attended with ulceration. In chronic cases, the mucous membrane becomes thickened and pigmented of a deep livid hue. Irregular ulceration occurs, and there is a deposit of triple phosphates on the surface. The muscular coat may hypertrophy ; and this is always the case when a stricture exists. There is at the same time a proliferation of fibrous tissue which leads to cicatricial contraction of the walls of the bladder.

The urine very often becomes purulent in acute cystitis. In chronic cases, it assumes a thick and gelatinous character owing to the action of the alkaline urine on the pus and other albuminous constituents of the inflammatory exudation. Microscopic examination of the urine reveals (1) pus corpuscles, (2) epithelium, (3) crystals of triple phosphate and (4) bacteria.

TUMOURS.

Simple.—The only tumour of importance is **papilloma.** It invariably grows from the base—where the urine is mostly in contact—and assumes the form of delicate villous processes (which are very prone to bleed) or a prominent warty mass. Other forms of tumour are occasionally found, and they all tend to assume a papillomatous form.

Malignant.—Various forms of **cancer** grow in the bladder. Primary cancer usually exhibits a villous character, and the villi are very liable to ulcerate and bleed. Secondary growths frequently occur by extension from the rectum or uterus.

PERFORATION

Of the bladder may occur under the following circumstances :—(1) Ulceration, usually the sequel of cancer, whether starting within the bladder or some adjacent organ. The irritation caused by a calculus may induce an extensive ulceration, resulting in perforation. (2) Rupture of a diverticulum. (3) A direct blow, sometimes accompanied by fracture of the pelvis. (4) Injury during parturition, leading to sloughing.

HÆMORRHAGE

From the bladder is the result of (1) cystitis, or (2) tumour growth, simple or malignant.

In this connection, it may be well to mention that hæmorrhage from the **kidney** occurs from the following causes:—(1) Acute nephritis; (2) calculi; (3) parasites, particularly the *Bilharzia hæmatobium*; (4) cancer.

DISEASES OF THE PERITONEUM.

ASCITES,

Or dropsy of the peritoneal cavity, is a very common result of obstruction in the portal circulation, arising from cirrhosis or cancer of the liver, or more rarely thrombosis of the portal vein. It also occurs as a part of general dropsy, caused by valvular disease of the heart, disease of the lung, and Bright's disease. It is evident then that the dropsy is a direct effect of increased transudation of fluid from the blood-vessels.

Characters.—The peritoneal sac is more or less distended with dropsical fluid, which gravitates to the lowest level of the sac, and rises higher and higher as it accumulates. It may ultimately fill the whole sac, unless in some cases where there has been previous inflammatory adhesion. The fluid possesses the usual properties of such effusions (*v.* page 18), but is occasionally milky from admixture of chyle, when it is found to contain flat globules and leucocytes. Further, when associated with cancer, typical cancer cells may be found in the fluid.

PERITONITIS,

Or inflammation of the peritoneum, is nearly always secondary to inflammation in other and neighbouring parts—in these organs in particular which lie within its folds. In some cases it arises apparently from exposure to cold, but the most common causes are (1) perforation of the bowel, induced by ulceration or gangrene; (2) operations on, and injuries to the abdomen; (3) extension of inflammation from the uterus during the puerperium. Acute peritonitis is often a special feature of the puerperal fever. A local peritonitis is frequently the result of inflammation in the ovary or Fallopian tubes.

Peritonitis may be *acute* or *chronic*.

Acute Peritonitis is almost always a septic condition, and rapidly proves fatal from septicæmia. In its anatomical details the condition is closely comparable to a purulent pleurisy or pericarditis, and the description given of the latter will serve in a general way for the other. It may be noted that the inflammation is always most intense, and the fibrinous exudation most copious wherever the coils of intestine are in apposition. The serous effusion is never so extensive as in a pleurisy. Further, the bowel is usually implicated in the inflammatory process. The wall of the gut becomes swollen and softened, there occurs a paresis of the muscular coat, leading to distension of the bowel by the pressure of its gaseous contents—a condition known as "meteorism."

Results.—(1) Recovery seldom takes place from a septic inflammation. Simple peritonitis may be cured by proper treatment. (2) Localized peritonitis is generally recovered from, but leaves its mark in the shape of chronic adhesions.

Chronic Peritonitis is generally a sequel of acute inflammation, and is in most instances localized. But it sometimes develops as a chronic condition in the course of Bright's disease, or in cases of tumour growth: under these circumstances the inflammation is a general one. The condition is characterized by the production of new fibrous tissue, and very often consequent adhesion between opposing surfaces. The thickening of the membrane which results from the new formation is most marked over such organs as the spleen and liver. The capsules of these organs may acquire a cartilaginous consistency, and it will be remembered that this is the origin of the so-called "flat fibroma." The adhesions are of greatest significance when they occur in the pelvis, because, in the first place, the intestine may become strangulated by their agency; and secondly, the uterus and its appendages may suffer displacement. Sometimes the pouch of Douglas becomes converted into a cyst by these adhesions, and its inflammatory contents may caseate and subsequently calcify. A local peritonitis has been known to cause occlusion of the foramen of Winslow.

TUBERCULOSIS

Sometimes gives rise to a **tubercular peritonitis.** This occurs as a primary disease, due to the specific action of the tubercular virus. It should be specially noted that tubercular ulcers of the intestine never give rise to a general peritonitis. The condition commences with a profuse development of tubercle granulations, which set up inflammation in the sac. The process becomes chronic, with the result that the peritoneum is greatly thickened and widespread adhesions occur. Amid the adhesions large caseous masses of tubercle are to be found, and in these typical giant cells can be observed. The omentum is frequently enormously thickened and contracted, so that it forms a huge solid mass in the upper part of the abdomen.

TUMOURS.

Primary tumours of the peritoneum are rare, with the exception of **cancer.** This is nearly always of the **colloid** kind, and forms great gelatinous masses in the cavity, matting all the parts together. This form

of cancer also occurs secondarily to cancer in the stomach or intestine, Certain kinds of **sarcoma** are occasionally met with as secondary growths in the peritoneum, for instance, **melanotic sarcoma** and **lympho-sarcoma.** A **fibro-sarcoma** is sometimes found in the omentum, and it grows to a very large size.

PARASITES.

Hydatids sometimes grow in the peritoneum. They are chiefly confined to the omentum, and in most cases are multiple. The **trichina spiralis** in the course of its migration from the intestine through the tissues sometimes lodges in the peritoneum, and gives rise to peritonitis.

DISEASES OF THE OSSEOUS SYSTEM.

OSTEO-MALACIA.

(SYNONYMS: "MOLLITIES OSSIUM"; "MALACOSTEON.")

This is a disease which is characterized by a general softening of the osseous tissue.

Characters.—The condition is usually first manifested in the bones of the pelvis, which are chiefly affected throughout the disease. At a later stage it extends to the vertebræ, ribs, femora, and other bones. The affected bones become so soft that they give way under the least pressure, and consequently deformity becomes a marked feature of the disease. The greatest deformity is perhaps produced in the pelvis, where the sides collapse and push forwards the symphysis pubis into a beaked process. This is due to pressure inwards by the heads of the femora, and is a frequent cause of difficult parturition, occasionally necessitating operative interference. The giving way of the vertebræ leads to spinal curvatures. The softened ribs may permit the chest to collapse, and so prevent full inspiration.

Morbid Anatomy.—There are two processes concerned in the disease—(1) decalcification, and (2) active cell proliferation and absorption. Decalcification takes place first in the cancellous tissue and in the central portions of long bones, and spreads towards the surface. "Rarely, however, does it attack the sub-periosteal outer lamellæ; in the most advanced cases, where the greater part of the bone may have disappeared, there nearly always remains a thin shell of comparatively healthy material which maintains the shape of the original bone." The decalcified bones are more vascular than normal, and hæmorrhages into the marrow frequently takes place. As the disease advances the quantity of marrow still increases, and is of a dull red colour.

The **microscope** reveals active cell proliferation of the bone marrow. Many of the cancellous spaces are densely packed with small cells; while in others giant cells are observed, their function apparently being to absorb the bony tissue.

Causation.—As to the origin of the disease almost nothing is known, but its occurrence is influenced by (1) old age; (2) insanity; and (3) pregnancy. "It is twelve times more frequent in females than in males, and among females more than two-thirds of the cases appear during the child-bearing period" (Greig Smith).

RICKETS (RACHITIS).

Rickets is essentially a disease of early life, rarely developing later than the second year. It generally commences between the twelfth and eighteenth month, never before the sixth month. Although the disease is most marked in the osseous tissue, yet it by no means limits itself to bones; the liver and spleen are also usually affected.

Characters.—The great deformity which rickets produces is partly due to softening of the bony tissue and partly to excessive growth at certain parts. The following points are note-worthy in the disease:—(1) Enlargement of the ends of the long bones, *i.e.*, at the line where ossification is proceeding, and in the parts which are about to ossify. (2) Softening of the bones. (3) Thickening of the flat bones, especially the bones of the skull, scapula and pelvis. (4) Deformity from mechanical causes acting on the softened bones. The general process giving rise to the above effects has been well described as an excessive formation of those structures which precede ossification, with retardation and great irregularity in the active process of ossification. There is an excessive development of the spongy tissue at the head of the bone, of the epiphysis, and of the layer of cartilage in which the primary deposit of calcareous matter takes place. At the junction of the epiphysis with the shaft the bluish cartilagenous lamina becomes very irregular in form and thickness. The bone which is formed is much more vascular and spongy than in the normal bone. The bones of healthy children yield about 37 per cent. of organic and 63 of earthly salts; whereas, according to Jenner, those of rickety children sometimes yield as much as 79 per cent. of organic and only 21 of earthy matter.

On **microscopic** examination one can see that the cartilage cells are crowded together irregularly, and many of them may be observed to undergo calcification. These cells may simply accumulate calcareous particles in their interior, or they may undergo a more complex process. The nucleus of the cartilage cell, which may or may not proliferate, is apparently transformed into a bone corpuscle lying in a lacuna. The cell itself may remain in an osteoid condition or completely ossify. There is thus formed a tissue between cartilage and bone, the great part of the structure being calcified cartilage. Along with this bone formation

a process of absorption takes place by osteoclasts, so as to form large spaces.

The general consequence of rickets is that the bones usually become arrested in development, giving rise to rickety dwarfs. The enlargement of the joint-ends of the long bones is especially seen at the wrist and ankle joints, and at the elbows and knees. Enlargements are also observed at the junction of the ribs with the costal cartilages, along the lines of the cranial sutures, and elsewhere where bones are in relation with epiphyseal cartilages. Curvature of the diaphysis of long bones is also a prominent condition in rickets. If the child walks much it becomes bow-legged. Should it be in the habit of sitting with the legs pendulous, an anterior convexity of the femur along with a sharpening of the posterior concavity results, giving rise to the so-called "sickle" shape. The ribs and sternal cartilages yield because of their softness, collapsing to some extent under the external atmospheric pressure, and the sternum is pushed forwards so as to form a "pigeon-breast." The effects of the disease on the pelvis differ according as the child is able to walk or not. If it be kept lying, the antero-posterior or conjugate diameter becomes greatly shortened; but, if the patient be able to walk, the antero-posterior diameter is lengthened, the mechanism of its production being the same as in the "malacosteon pelvis." Curvatures of the spine also occur. The cranium sometimes becomes greatly thickened, enlarged and square shaped; but on certain parts of the skull the bone is very thin, there being little else but the periosteum and dura mater to protect the brain—a condition called **craniotabes**.

Causation.—The disease has been said to be due to an excess of lactic acid in the blood. As the condition is almost exclusively limited to children of the poorer classes, and is especially common in large towns, there is no doubt that defective hygienic conditions favour its occurrence. Children brought up by the hand and who receive a diet unsuited to their age, are specially prone to the disease. M. Guérin, in his experiments, removed puppies from their mother, weaned them at a very early age, and fed them on raw flesh (an unsuitable diet at an early age), with the result that in four or five months they were rendered rickety. An excessive use of starchy foods is also an important factor in producing rickets. (Greenfield).

OSTEITIS DEFORMANS (PAGET).

This disease has been confounded with osteo-malacia. It is a rare condition occurring in old age, and consists in increase of bulk and distortion of the bones. The bones first affected are the tibia, clavicle or skull bones. "They become enlarged in all their dimensions, rarefied in their compact proportions, and thickened and roughened under the periosteum." On section the medullary canal is seen to be narrowed as a result of the thickening, but the bone is much more spongy than normal and is more uniform in structure, not showing any superficial compact bone. The thickening of the skull is often enormous, reaching to an inch sometimes. The head in consequence is enlarged and is usually asymmetrical. Distortion in shape of the spine, pelvis, thorax and skull clinically mark the disease.

TUBERCULOSIS OF BONES AND JOINTS.

This condition includes the majority of cases of **caries**, **white swelling** of joints, **chronic synovitis**, and **strumous arthritis** (Greenfield). The tubercular process may begin in the bone itself or in the periosteum. In the case of joints it commences in the synovial membrane or in the bone at the epiphyseal line. The disease is sometimes very chronic, and may be limited to one bone or joint. As regards the bones and joints affected, the most frequent of the former are the vertebræ, tibia, pelvis, ribs, tarsus and carpus; of the latter, the hip, knee, ankle, tarsal, and carpal joints are the commonest.

Characters. I. Bones.—When tuberculosis attacks bone it usually commences with medullary tubercle granulations in the cancellous tissue. Typical giant-cell systems make their appearance early; they increase in number and size and set up two sets of changes:—*(a)* Inflammatory—proliferation of the marrow, increased vascularity and the absorption of fat. The osteoclasts are also increased in number. *(b)* Necrotic—under the influence of the osteoclasts, the trabeculæ disappear and the bone becomes extremely rarefied. Caseation and necrosis subsequently set in, and we may get a large abscess cavity formed.

When the periosteum is primarily affected, the deep layer shows the first change. Typical giant celled granulations may be observed, the subsequent changes being similar to those studied in bone.

II. **Joints.**—The synovial membrane is irregularly thickened and gelatinous, there are numerous outgrowths on the synovial fringes, and at the periphery of the cartilages there are irregular granulation masses. The cartilage is usually softened, and at parts eroded. The surrounding tissues are usually much swollen. The articular ends of the bones are very soft, and may undergo caries. If the case be far advanced, there may be very great destruction of the bone.

Microscopically.—The first change is observed in the synovial membrane, the margins of which are seen to be greatly thickened and covered over with an irregular fungating mass. Towards the surface there is an amorphous substance consisting of broken down cells, while a little deeper are masses of granulation tissue, of which the more superficial are tubercular. In the cartilage, areas of softening can be observed, and granulation tissue may be traced into it from the synovial membrane. Giant cells, however, are not seen. In the tissues surrounding the joint

there is scattered granulation tissue, perhaps with tubercle here and there.

Although sometimes we can trace a direct extension of the tubercle bacillus from an internal organ to a bone or joint, *e.g.*, from the mediastinal glands to the ribs or sternum, or from the supra-renal capsule to vertebræ, yet frequently it is quite uncertain how the virus gets to the part. It is highly probable that the organism travels by the blood or lymphatics in such obscure cases, or by both. This local tuberculosis is often preceded by a blow or injury to the part.

SYPHILIS.

Bone disease, the result of acquired syphilis, is a manifestation of the tertiary period. It tends especially to affect the deeper layers of the periosteum, causing localized collections of small celled inflammatory material. These may be regarded as gummatous infiltrations. They are soft and may fluctuate, and not unfrequently go on to suppuration and abscess formation. If the suppuration detach a great part of the periosteum from the bone, the latter, being deprived of its nutrition, will undergo necrosis. The bones which are chiefly affected in syphilis are the tibia, ulna, clavicle, sternum, ribs, and those of the cranium (especially the parietal and occipital).

RHEUMATIC ARTHRITIS.

This condition usually follows an attack of acute rheumatism, although a similar affection (osteo-arthritis deformans) takes place in old people. The synovial membrane shows an irregular thickening, and vascular outgrowths appear from the margins. These outgrowths may become detached and form loose bodies in the joint. The articular cartilage undergoes degeneration, it softens and becomes eroded along the lines of pressure.

DISEASES OF THE NERVOUS SYSTEM.

I. BRAIN and MEMBRANES.

MALFORMATIONS.

I. **Anencephalia,** or absence of the encephalon. When the absence of the brain is not complete, the deficiency is usually most marked in the part corresponding with the cerebral hemispheres; portions of the ganglia at the base and the cerebellum remaining. The condition is incompatible with life.

II. **Microcephalus,** or an abnormally small brain, is usually attended with defective development of certain convolutions. It is always accompanied with more or less idiocy.

III. **Encephalocele** is a protrusion of the brain substance through some opening in the skull. The condition is therefore associated with

IV. **Meningocele**, which consists in a protrusion of the meninges only.

V. **Hydrencephalocele** is the term applied when the protruded mass of brain tissue is generally distended with fluid from a dropsical ventricle. Of the three conditions last mentioned, this is the commonest.

These protrusions are due to congenital defects in the development of the skull, whereby gaps are left in the cranial bones. The condition is most frequent along the lines of the fissures, either posteriorly in the occipito-parietal region or anteriorly in the frontal.

INJURIES.

Recent advances in surgery have shown that a large portion of brain substance may be removed without serious results. When a portion of the skull is removed by injury or operation, and an aperture made in the dura mater, we may get what is known as a **hernia cerebri** formed. In this condition a portion of the brain substance protrudes. It seems to arise from the removal of the natural pressure of the bone.

Hæmorrhage into the brain and its membranes may result from direct or indirect injury. It may take place *(a)* outside the dura mater, between it and the skull, detaching the dura and producing effects by compression of the brain ; *(b)* into the sub-dural space. The hæmorrhage may take place from the dura mater or from the brain substance itself. As the inner layer of the dura mater is much less vascular than the outer, it is probable that unless the dura be actually torn through, hæmorrhage into the sub-dural space results from laceration of the brain or piameter. The effused blood usually gravitates to the base of the brain. Blood extravasated outside the dura accumulates and undergoes changes leading to "*pachymeningitis hæmorrhagica.*"

Contusion of the brain is usually the result of some injury to the skull. The effects which are produced on the individual are clinically known as **concussion**. Concussion may be briefly defined as "surgical shock with the additional brain symptoms."

When the head is exposed to any serious blow—whether directly, or indirectly through the trunk—the consequence is a partial laceration of the brain, and this occurs in two positions :—(1) At the point where the blow is given ; but the more serious results are got at (2) the opposite pole of the encephalon. Thus, if a blow be struck on one side of the forehead the contusion will be especially found over the temporo-sphenoidal and occipital lobes of the opposite side. Again, if the injury be over the vertex posteriorly, the contusion is more marked at the tips of the frontal lobes. This form of injury is known as "*contre coup.*" If an

arachnoid mater ?

injury be inflicted over a wide area the part struck may not be injured at all—only the opposite pole.

A. MEMBRANES of the BRAIN.

MENINGITIS.

I. PACHYMENINGITIS.

This essentially consists in an inflammation of the dura mater, the dura arachnoid being less affected.

Sites.—The condition may occur at any part, but it is most common over the vertex, owing to its exposure. When it follows disease of the ear, *e.g.*, tubercle of the middle ear and of the mastoid antrum and cells, the inflammation affects specially the middle and posterior fossæ at the base.

Characters.—The membrane becomes swollen and highly injected, then usually turns dark livid or green in colour. This change of colour is due to decomposition of blood. The dura mater often becomes detached from the bone, and an abscess may form between. The disease is usually more or less associated with suppuration. The bone becomes roughened, infiltrated with pus, and then necrosed. Microscopically the phenomena are the same as those seen in inflammation of any other fibro-serous membrane.

Causes.—(1) Traumatic injury to the skull or dura mater. (2) Secondary to chronic disease of the bone, *e.g.*, of the ear. (3) Secondary to tumours and gummata of the bone or of the external surface of the dura itself.

Thrombosis of the Cerebral Sinuses.

Causes.—(1) Pachymeningitis. (2) Marasmus, *i.e.*, extreme prostration through disease or loss of blood. This is the most frequent cause of thrombosis in the longitudinal sinus, which may come to be filled throughout its whole length by a firm clot. (3) Disease in the middle ear is the most common cause of thrombosis in the lateral sinus, extending to it from the posterior aspect of the petrous bone. But the lateral sinus may also be affected by extension from the petrosal sinus. The cavernous and circular sinuses are liable to become thrombosed in consequence of (4) tumours involving the sella turcica and (5) syphilitic disease of the membranes in that position.

Results.—By producing great engorgement of the superficial veins, thrombosis of the sinuses frequently gives rise to hæmorrhage. If the person survives, the thrombus may become organized. Following disease of the ear thrombosis is septic. The clot undergoes decomposition and softening, and the phlebitis which is set up is apt to spread to the jugular veins and cause death by septic embolism.

II. LEPTOMENINGITIS.

This consists of an inflammation of the pia mater. It may be said as a general rule, that all traumatic and surgical meningitis is pachymeningitis; while all medical meningitis is leptomeningitis.

Sites.—The condition may occur (1) on the vertex, (2) at the base, (3) in localized patches. Primary leptomeningitis which is not tubercular is usually found on the vertex. Leptomeningitis of the base is almost invariably tubercular or is secondary to meningitis of the cord. Localized leptomeningitis is usually due to disease spreading from the dura mater or brain.

Characters.—In the early stages the pia mater is vividly injected, and becomes of a rosy red colour; not simply venous fulness. The condition spreads into the superficial part of the brain; and minute hæmorrhages may take place. Hæmorrhage, however, more frequently takes place in *epidemic cerebro-spinal meningitis* and *septic meningitis* following ulcerative endocarditis. As a result of the inflammatory process, the sub-dural space becomes filled with fluid, which is at first turbid and opalescent, and later yellow and slightly grey in colour. When meningitis starts from the dura mater there is a large quantity of exudation between the dura and anachnoid. But when the inflammation begins in the pia mater the exudation is small in amount, as it is held in the meshes of the arachnoid.

Microscopically.—The vessels are engorged with blood, there is much fibrinous exudation in the meshes of the pia mater, and leucocyte emigration is seen to take place along the delicate vessels which pass into the substance of the brain.

Causes.—Leptomeningitis may arise from the following causes:—(1) secondary (a) pachymeningitis, (b) inflammation from outside, *e.g.*, from the eye by way of the sheath of the optic nerve, (c) inflammation of the membranes of the spinal cord, (d) extension from the brain substance; (2) primary, (*a*) inflammatory softening of the brain, *e.g.*, round a tumour which is usually localized, (*b*) causes which give rise to inflammation of other serous membranes, *e.g.*, septicæmia, pyæmia, acute pneumonias; (*c*) acute fevers, measles, whooping cough, small-pox, etc., (*d*) epidemic cerebro-spinal meningitis, (*e*) general conditions which produce pleurisy and pericarditis — acute rheumatism, Bright's disease, etc. (*f*) tuberculosis.

III. TUBERCULAR MENINGITIS.

This condition, which is also known by the name of "**acute-hydrocephalus**" is a very common disease among children. The later name is applied on account of it producing distention of the sinuses. It is usually an accompaniment of general acute tuberculosis, but it may be the only manifestation of tubercle.

Sites.—The disease always commences at the base of the brain, starting in the region of the inter-peduncular space and spreading upwards along the lines of the great fissures towards the vertex. Very frequently it arises secondarily by extension from the cord.

Characters.—A tubercular infiltration is found along the perivascular sheaths of the pia mater, which gives rise to a varying degree of inflammation. The infiltration is chiefly at the base of the brain, and is seen to extend along the fissures towards the vertex. On the upper surface the hemispheres are found bulging, usually unequally, and the convolutions over the vertex are flattened. The lateral ventricles are distended with turbid serum. On section, one can observe that the parts of the brain round the ventricles are softened. The pia mater is found to undergo inflammatory changes similar to those already described.

Microscopically. — Along the small vessels elongated fusiform swellings may be observed. They consist of masses of cells in the perivascular sheath; each cell being irregular in shape and having two or more nuclei. These are the early stage of tubercle structure, but it is rare to find the complete giant-cell system.

B. The BRAIN SUBSTANCE.

ANÆMIA.

I. General anæmia of the brain occurs in many wasting diseases, as well as from copious loss of blood. II. Local anæmia is usually due to vascular obstruction. The **causes** may be (1) embolism, most commonly a result of disease of the cardiac valves; (2) thrombosis in the arteries, the two most common varieties being *(a)* atheroma and *(b)* syphilitic disease.

Characters.—These vary according to the degree of the anastomosis of the vessel affected. If the obstructed vessel be a terminal one, or its anastomotic power small, softening of the brain substance sooner or later ensues. At first, the area supplied by the affected vessel becomes hyperæmic, and even minute hæmorrhages may be observed. Very soon, however, pallor succeeds, and softening follows. The brain substance then comes to be yellow in colour and breaks down into a pulpy mass. This mass consists of broken down and fattily degenerated elements, fat and cholesterine crystals. After a time the necrosed tissue may become completely absorbed, leaving a milky fluid. The presence of the degenerating tissue gives rise to irritation of the surrounding substance which results in a chronic inflammation of these parts. If the condition takes place on the surface of the brain the pia mater becomes thickened and adherent to the brain and dura mater: especially is this the case at the base of the brain.

Sites.—Embolism most commonly affects the middle cerebral arteries, the left being especially affected because of the more direct course of the left carotid from the aorta. Thrombosis when due to atheroma is more common in the branches of the posterior cerebral, and in the basilar arteries than elsewhere. Syphilitic thrombosis may occur in any of the vessels.

INTRA-CRANIAL HÆMORRHAGE.

I. MENINGEAL HÆMORRHAGE.

Hæmorrhage may take place outside the dura mater, into its substance or beneath it—subdural. Subdural hæmorrhage is supposed to be the cause of **pachymeningitis hæmorrhagica.** This condition, which is also known as "hæmatoma of the dura mater," consists in a blood clot covering the internal surface of the dura mater, and compressing the brain substance. The blood, which varies in quantity, is contained between walls formed by a thickened portion of the dura. Pachymeningitis hæmorrhagica is most frequently found over the vertex, and there are two views as to its production. According to one view a hæmorrhage takes place into the arachnoid cavity, and this becomes encapsuled by a false membrane, and adheres to the dura mater. According to Virchow the disease begins in a chronic inflammation of the dura mater. The condition is found in chronic alcoholism and insanity.

II. CEREBRAL HÆMORRHAGE.

By this term is meant bleeding into the substance of the brain. The causes of cerebral hæmorrhage may be enumerated as follows :—(1) Traumatic injury ; (2) diseases of the blood vessels—aneurism, atheroma and fatty degeneration ; (3) blood diseases, such as leucocythæmia, scorbutus and certain fevers. In blood diseases hæmorrhage usually takes place by diapedesis, but in other cases there is always rupture of one or more vessels. By far the commonest cause ef cerebral hæmorrhage is aneurism of the arteries.

Aneurisms of large size are usually situated on the circle of Willis or some of its larger branches. They are most common on the anterior and middle cerebral arteries. Unlike aneurisms in other positions they may grow at the age of twenty or thirty, and sometimes co-exist with valvular disease of the heart. They produce two sets of changes,—(1) pressure as in the case of tumours ; (2) rupture. The commonest form of cerebral hæmorrhage is into the substance of the brain, and in about 90 % of cases it takes place in the region of the basal ganglia. This is probably because the vessels supplying these parts come from the middle cerebral artery, and have a more direct course from the heart so that they are under great pressure. Hæmorrhage from rupture of a vessel especially occurs in connection with chronic Bright's disease and hypertrophy of the left ventricle. Miliary aneurisms, which are usually multiple, occur as fusiform or sacculated dilatations from $\frac{1}{40}$ to $\frac{1}{12}$ of an

inch in diameter. If the hæmorrhage be not fatal the effused blood undergoes changes such as are found in any ordinary sanguineous effusion. After absorption of the blood clot, however, the yellow fluid which remains sets up a chronic irritation in the brain substance which becomes condensed, and forms a cyst encapsuling the residue of the clot. This cyst contains hæmatoidin crystals, which distinguish it from a cyst formed from softening of the brain substance.

ENCEPHALITIS.

Inflammation of the brain may be either acute or chronic.

I. ACUTE ENCEPHALITIS.

We have already seen that in the midst of hæmorrhagic foci and round tumours there are inflammatory manifestations. The result of these changes is the formation of fibrous tissue, constituting a cyst or a cicatrix. But if the brain has been injured traumatically so that the brain substance communicates with an external wound, suppuration takes place. Laceration of the brain may take place without any external wound or fracture of the skull, as by a fall on the head.

Abscess formation may be the ultimate result of encephalitis, but this probably is due to the introduction of some septic matter. Such abscesses are usually chronic, and may form large cavities. They contain thick pus with a very fœtid odour, swarming with bacteria, and often containing blood. They usually have a distinct firm capsule, which is composed of inflammatory tissue. The capsule may be considered as sclerosed brain tissue. Outside the capsule the brain substance is, as a rule, more or less softened. Abscesses of the brain may arise from (1) pyæmia. Pyæmic abscesses are usually associated with ulcerative endocarditis; (2) septic conditions of other organs such as the nose, the antrum of Highmore, the orbit, and the middle ear; (3) traumatic injury, when the condition allows of the introduction of septic matter.

II. CHRONIC ENCEPHALITIS (CEREBRAL SCLEROSIS).

There are three different varieties of chronic encephalitis described

1. DIFFUSE SCLEROSIS.

This condition is supposed to be due to some irritant circulating in the blood, and therefore one might expect it to attack all parts equally. In delirium tremens, tubercular meningitis, uræmia, erysipelas, etc., the brain is over-run with leucocytes; and the irritation of these cells in supposed to set up diffuse inflammation in the brain substance.

General paralysis of the insane.—This condition may be considered as one form of the disease. The morbid anatomy of the disease may be briefly summed up in the following points. The dura mater is usually thickened and adherent to the skull. The pia mater also exhibits thickening in the form of milky patches; and it is adherent to the surface of the convolutions, so that on attempting to remove it, bits of the brain substance come away. The convolutions are atrophied in certain regions, and the sulci appear abnormally filled with fluid. The brain is greatly reduced in weight though it may be firmer than normal.

Microscopically.—Around the small arteries in the pia mater and brain substance there is proliferation of the neuroglia and little clumps of pigment, the remains of former extravasations. The ganglion cells are shrunken and often pigmented, and the blood-vessels are found to have undergone fatty degeneration.

2. *DISSEMINATED SCLEROSIS.*

This disease essentially consists in patches of chronic inflammation scattered throughout the brain substance. These patches occur most commonly in the white matter, and are usually found towards the base. They produce induration and shrinking of the brain substance. " Under the microscope the sclerosed white substance shows loss of the medullary sheaths of the fibres, while the axis cylinders, according to Charcot, are largely retained."

3. *SYSTEMATIC SCLEROSIS.*

This is usually the result of a destructive lesion involving some of the conducting tracts in the brain. A sclerosis of the motor part of the internal capsule is sometimes found, which consequently causes "descending degeneration" along the pyramidal tracts. (See Cord).

HYDROCEPHALUS

Consists in a mechanical distention of the brain substance, due to an accumulation of fluid in the ventricles.

I. **Acute hydrocephalus** as we before saw occurs in tubercular meningitis. It is probably of inflammatory origin.

II. **Chronic hydrocephalus** may be *(a)* acquired or *(b)* congenital.

(a) The acquired variety usually occurs late in life, and as a rule is the result of pressure from a tumour. Most frequently the growth occurs in the middle lobes of the cerebellum, obstructing the veins of Galen. Pressure upon the straight sinuses might also give rise to the condition.

(b) Congenital hydrocephalus is usually found at or shortly after birth, and gradually increases in intensity. It is distinguished from the other form by dilatation of the whole head. The ventricles become enormously distended with fluid, and as a consequence the brain substance is expanded over them. The brain, as a result of this pressure, atrophies. The pressure ultimately tells on the bones of the skull which undergo great distention. The fissures and fontanelles become widely separated, and in the intervening membranes small plates of bone are formed. The enlargement of the head takes place transversely and

antero-posteriorly, and the orbital plate is depressed and rendered more oblique, causing the child's eyeballs to protrude.

CHRONIC TUBERCULOSIS.

Chronic tubercle is most frequently met with in the cerebellum. In the cerebrum the tubercles grow in the cortex, and are usually multiple. They are rounded and laminated masses, consisting of two or three centres of growth. Towards the outer part they are generally firm and sharply defined and have an apparent translucent capsule: the interior is yellow and caseous.

Microscopically.—The nodules show typical tubercular structure. There is an internal caseous part, and a peripheral part composed of tubercular systems.

Chronic caseous tubercle usually acts on the surrounding brain substance like a cerebral tumour, producing inflammation and consequent softening.

SYPHILITIC DISEASE OF MEMBRANES AND BRAIN.

(1) The **dura mater** is by far the most frequent seat of intracranial syphilis. The disease usually occurs in the form of gummatous infiltrations, and is most frequently found on the vertex. When the condition involves the outer aspect of the membrane, the disease usually involves the bones of the skull, either from direct pressure or infiltration: if, however, the internal surface of the dura mater be chiefly affected the brain is apt to suffer. These gummata may form large tumour masses and cause atrophy of the surrounding structures.

(2) The **pia mater** is less often the seat of syphilitic changes. It is frequently affected secondarily to the dura, when the two membranes become adherent. Gummata in the pia mater are usually of small size and soft consistence.

(3) In the **brain** gummata are as a rule secondary to syphilitic growths elsewhere. They may be deeply seated in the brain substance, but more frequently they are on the surface. They may reach the bulk of a hen's egg.

(4) It is not uncommon for the **cerebral arteries** to be affected in syphilitic disease. Indeed, they are always more or less affected when syphilitic changes are taking place in the pia mater, although the arteries may be affected apart from the latter. The usual condition is to find inflammatory changes in the outer coat along with endarteritis obliterans. The arteries chiefly affected are the main branches of the circle of Willis or those running over the surface of the brain.

INTRA-CRANIAL TUMOURS.

The more rare tumour growths found within the cranium are the

fibroma, psammoma, melanoma and the **lipoma**. The following are of more frequent occurrence :—**myxomata**, which may be found either in the membranes or the cerebral substance ; **gliomata**, growing in connection with the nerve centres ; **sarcomata** may grow either in connection with the dura mater or brain proper.

Effects—Intra-cranial tumours produce their effects partly by pressure and partly by invasion of the brain substance. The fatal result is caused by direct pressure on a vital part or acute inflammatory softening around the tumour.

PARASITES.

Cysticercus cellulosæ and the **hydatid** are the only parasites which infest the brain of man. The former are most commonly found in the cerebral hemispheres. The hydatid cyst when found in the brain is usually barren and solitary, and is associated with hydatids in the liver or other organ.

II. SPINAL CORD and MEMBRANES.

HYDRORACHIS.

By this we mean an accumulation of cerebro-spinal fluid either in the central canal or arachnoid cavity of the spinal cord, resulting in a sacculated distention of the cord and its membranes. It is generally a fœtal condition, and is nearly always associated with a congenital deficiency of certain of the vertebræ, in consequence of which the soft tissues of the back are stretched over the protruding sac. This gives rise to a fluid tumour or cyst situated somewhere in the middle line of the back, constituting what is known as **spina bifida.**

There are two varieties of hydrorachis, distinguished as **h. interna** and **h. externa.** In the first variety, the fluid accumulates within the central canal of the cord, stretching and thinning the nerve matter; ultimately the cord gives way posteriorly, and the fluid is extravasated into the arachnoid cavity. The wall of the tumour thus comes to be composed of (1) pia mater, (2) arachnoid, (3) dura mater and (4) skin and integuments. In the second variety, the fluid is confined to the arachnoid cavity, and there is no communication with the central canal. In this case the sac of the tumour is composed mainly of (1) thickened dura mater, supported by (2) the skin and integuments. In both varieties, the condition might be aptly considered as a hernia of the spinal membranes.

Spina bifida usually occurs in the sacral or lumbar region, rarely higher. An examination of the vertebræ in the affected region shows that the neural arches are more or less deficient. The spinal cord is not as a rule displaced, but its lower extremity may be involved to some

extent in the cystic process. This applies particularly to h. interna. Thus, the filaments of the cauda equina are dragged into the sac and become fused with the sac wall; while the filum terminale often has a point of attachment to the interior of the sac and produces a depression or dimple on the outer aspect of the tumour.

MENINGITIS,

Or inflammation of the spinal meninges, may be classified in the same manner as inflammation of the cerebral membranes.

I. PACHYMENINGITIS.

A. **Pachymeningitis externa** is in most cases a tubercular affection, and as such falls to be described under "Tubercular Meningitis." The thickening of the dura leads to pressure upon the cord and upon the nerves as they emerge through their respective foramina; and very frequently the process extends to the deeper structures.

A **simple** pachymeningitis externa may result from injuries to the spinal column. A **suppurative** inflammation is not uncommonly the sequel of a bedsore over the sacrum; such a bedsore sometimes ulcerates so deeply as to reach the vertebræ and set up inflammation in the dura. The result is an extensive suppuration outside the dura, which appears swollen, opaque, and of a dark livid colour. The inflammation may extend inwards to the other membranes.

B. **Pachymeningitis interna** is caused either by tuberculosis or by syphilis. There are two varieties of the condition:—(1) **P. Hypertrophica.** This occurs especially in the cervical region, and is manifested by a laminated thickening of the dura in the shape of concentric rings of fibrous tissue, encroaching on the pia mater and arachnoid, and matting all the membranes together. The spinal cord, in consequence, is compressed, as well as the nerves at their points of exit from the canal. (2) **P. Hæmorrhagica** is similar to the condition of the same name occurring in the cerebral dura. It may be either of spontaneous or traumatic origin.

II. LEPTOMENINGITIS.

This signifies inflammation affecting the arachnoid and pia mater of the cord. It occurs under the same conditions that give rise to cerebral meningitis, and is often associated with it: thus it may be simple or tubercular. A simple leptomeningitis is usually most marked in the pia mater, being the more vascular structure. It becomes congested, and exhibits minute ecchymoses. The arachnoid also shows congestion, and the cerebro-spinal fluid becomes turbid. Inflammatory exudation is poured out, and the contents of the arachnoid become purulent. This purulent infiltration is found most abundantly on the posterior surface of the cord, a circumstance due to the action of gravity dependent on the dorsal position of the patient. But, further, the exudation is frequently most marked over the dorsal and lumbar enlargements, an unaccountable fact. The dura often becomes secondarily inflamed, as also the cord.

III. TUBERCULAR MENINGITIS.

This very often occurs in **chronic** form as a sequel to caries of the vertebral column, and affects the dural sheath mainly. The dura becomes greatly thickened over an irregular area, and the spinal canal is filled up with a mass of caseous material, which unites the dura to the bone. The thickening of the dura leads to pressure upon the nerves as they emerge through their respective foramina, and very frequently the process extends to the deeper structures. The inner surface of the dura mater is especially affected in the cervical region, and this phenomenon is associated with caries of the atlo-axoid articulation. When the dura is opened into, it presents a gelatinous thickening covered with tubercle granulations.

An **acute** form of tubercular meningitis occurs in the course of acute tuberculosis. It nearly always exists along with cerebral tubercular meningitis, and very probably tubercular meningitis of the brain often spreads up from the cord. The morbid appearances produced are very similar to those of leptomeningitis, with these differences, that the effusion is less plentiful but more purulent, and tubercle granulations are seen scattered over the arachnoid.

HÆMORRHAGE.

The cord and membranes are seldom the seat of hæmorrhage, still it occurs under certain conditions. In fractures of the spine, or violent injuries to the back, bleeding sometimes takes place into the spinal canal, external to the dura mater. In rare instances one finds hæmorrhage within the arachnoid cavity: this can generally be traced to a lesion in the base of the brain or medulla, where blood has been extravasated and found its way down the cord. This "intra-meningeal" hæmorrhage is most liable to occur in some forms of insanity. Blood effused into the spinal canal may produce pressure effects on the cord.

EMBOLISM.

Embolism of the cord is chiefly known in connection with experiments on animals; it is rarely observed in man. Artificial embolism produces small points of localized softening, bounded by a hyperæmic zone of healthy tissue. Possibly the occurrence of myelitis may be in some way related to capillary embolism or to thrombosis.

MYELITIS.

By this term we mean an inflammation or softening of the substance of the spinal cord, acute or chronic.

I. ACUTE MYELITIS

Usually occurs secondarily to some other condition, and the following causes may be enumerated :—(1) Traumatic injury, from fracture, dislocation, or curvature of the spine. (2) Pressure of tumours. (3) Extension of inflammation from the meninges. (4) Fevers. In some cases acute myelitis occurs from simple exposure to cold when the body is overheated.

When any damage affects the cord externally the inflammation tends to be localized at the point of injury, where it involves the whole thickness of the cord. This gives rise to the specialized condition of **transverse myelitis.** In many cases of myelitis the inflammation is localized in a different manner, being limited (as to its breadth) to one or more of the nerve tracts in the cord, and extending a varying distance up or down the cord. This condition is spoken of as **systematic myelitis.** On the other hand, we meet with some forms of inflammation where extensive and unlimited portions of the cord are involved, and here we have to deal with **diffuse myelitis.**

Characters.—In an early stage the cord is injected, showing a red mottling on the surface and capillary hæmorrhages. The inflamed part swells and becomes quickly softened. The hyperæmia then disappears, the cord becomes yellow or yellowish-white and is converted into a mass of pulp, which oozes out of the membranes on making a section.

Microscopically, the pulp mass is found to consist of globules of myelin and "compound granule globules," the latter being altered neuroglia cells, with leucocytes scattered here and there. At a later period, slow absorptive changes occur, the cord shrinks and becomes largely replaced by connective tissue. The shrinking may be so great that the cord is only one fourth of its normal diameter.

The inflammation is liable to spread, and extension mainly takes place upwards. Further, the condition may become chronic, and be followed by ascending and descending degeneration.

II. CHRONIC MYELITIS.

This term indicates a chronic inflammatory or degenerative process in the spinal cord, attended with fibrous tissue formation. When of inflammatory origin, the disease may be a sequel of acute myelitis or meningitis, or it may partake of a chronic character from the beginning. The most frequent cause is a slow compression of the cord, as by a tumour or a curvature of the spinal column. But there is a form of chronic myelitis which may be described as a degeneration of the nerve elements with a secondary overgrowth of fibrous tissue, and which sometimes goes by the name of "parenchymatous degeneration." In both of these forms the morbid phenomena are very similar.

Characters.—Chronic myelitis leads to changes in the cord which are commonly indicated by the term "sclerosis." (This is equivalent to "cirrhosis" in other organs.) When any part becomes sclerosed, the degenerated tissue appears more grey and translucent than normal, and (unlike healthy tissue) takes on a very faint stain with osmic acid. On the other hand, carmine and logwood impart a deeper stain than natural. The cord is usually shrunk in some degree.

Microscopically, the medullated nerve fibres are irregularly swollen: and the medullary sheath breaks up into a mass of globules, which are absorbed. The axis cylinders show a series of spindle-shaped swellings, and they stain very deeply with certain reagents. Gradually the cylinders suffer disintegration like the medullary sheath, and are absorbed, though at a later period than the other. The empty nerve sheaths persist for a long time. Simultaneously with these changes we find others. The cells of the neuroglia enlarge; the connective tissue fibres swell, and become coarser and denser: thus there is formed a dense fibrillated tissue, in which lie many branching cells, mixed with small round cells—probably leucocytes, exuded from the blood-vessels. In the grey matter certain special phenomena are observed. The cells in the anterior cornu swell and show an increase of pigment: their processes break off, and the cells then atrophy and disappear. The neuroglia in the grey matter proliferates and forms a delicate fibrillated network supporting large branching cells, the structure resembling that of a glioma. The blood-vessels exhibit inflammatory changes, and one often observes around them cyst-like spaces in the nerve tissue, due, apparently, to the unequal contraction of the fibrous overgrowth.

The changes in the grey matter are usually attended with shrinking, and this leads to more or less deformity of the cord. This condition is found in a variety of diseases, and it is usual to group the diseases under certain types of sclerosis.

I. **Disseminated Sclerosis.**—This resembles the condition of the same name in the brain. The cord may be affected alone, but the brain is often associated in the disease—in fact, in all cases where the brain is affected the cord is involved along with it.

Characters.—The degeneration occurs in patches here and there towards the surface of the cord, and limited to the white matter as a rule. The patches have a tendency to local increase, and so may come to involve the grey matter; but further, when they are situated on any of the conducting paths, the latter undergo secondary degeneration.

II. **Secondary Degeneration.**—Suppose the cord to be damaged at a certain point by inflammation or pressure, what is the result? Take the case of caries of the vertebræ. The damaged part of the cord undergoes inflammatory softening in its entire extent, *i.e.*, a transverse myelitis occurs. But above and below the seat of softening, we observe secondary changes involving particular paths in the cord and extending some dis-

tance in the longitudinal axis. Let us now trace these changes along the cord, a proceeding in which we are guided by the grey gelatinous appearance of the degenerated parts. Going down the cord, we find a degeneration affecting the lateral columns chiefly, corresponding to the crossed pyramidal tracts: this is best seen high up, it becomes more limited in its area the lower we go, and may disappear in the lumbar region. The direct pyramidal tract in the anterior column is also affected, but the fibres of that tract being short, the traces of degeneration soon disappear. Passing up the cord now, we first observe changes affecting the whole of the posterior columns, including the posterior median column and postero-external column, as well as the direct cerebellar tract; further up, the posterior median columns and direct cerebellar tract are alone affected, and it may be possible to trace the degeneration in these tracts up to the restiform body and cerebellum respectively. The lateral column is also involved to a slight extent.

There is a certain number of degenerations in the cord which are secondary to a lesion in the brain. If the motor part of the internal capsule suffer damage, or if from any cause (whether tumour, hæmorrhage or softening) the motor tract in the brain be rendered unable to conduct motor impulses to the cord, there ensues a secondary degeneration in the cord extending along the paths of normal motor conduction and perhaps reaching to the limits of the cord.

DISEASES ASSOCIATED WITH SECONDARY DEGENERATION.

I. **Locomotor ataxia.**—The change found in the cord in this disease is mainly limited to the posterior columns. It commences in the lumbar region, close to the central canal; travels up in the posterior median columns, invading the columns of Goll; and in the cervical region diminishes in its lateral extent, so that there the columns of Goll are alone affected, the sclerosed parts being close to the median fissure. Together with this we usually find degeneration of the posterior root zone, and these roots are consequently much atrophied.

II. **Spastic paralysis** is a disease in which the characteristic lesion is lateral sclerosis. It usually commences in the lumbar region, and attacks both pyramidal tracts, afterwards proceeding along the cord. Usually, there is a degree of lateral extension, and the ganglionic cells in the anterior cornu are invaded.

III. **Infantile spinal paralysis.**—Here there is a descending sclerosis specially affecting the anterior cornua, and as the disease is an acute one it is commonly designated by the synonym of "poliomyelitis anterior acuta." The disease is usually associated with acute meningitis of the cord. The ganglion cells in the anterior cornu atrophy, and so the cornu shrivels. Ultimately it becomes limited to certain regions, and it produces atrophy of certain groups of muscles.

IV. **Progressive Muscular Atrophy** is another disease associated with atrophy of the ganglionic cells in the anterior cornu of grey matter. It is characterized by an atrophy commencing usually in the muscles of the ball of the thumb or little finger, and gradually extending over the body.

SYPHILIS

Of the cord and membranes has been less frequently observed than in the brain, but the changes are similar. It starts in the dura mater, and causes secondary changes in the cord, leading to sclerosis.

TUMOURS.

The great majority of tumours in the cord are secondary, starting from the bones of the spinal canal or extending from distant parts. **Glioma**, **psammoma**, **fibroma**, and **sarcoma** have all been found.

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